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A METHOD OF TREATMENT OF PURULENT SINUSITIS

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This treatment consists of the use of indwelling polythene tubing in the antrum for a prolonged period for the purpose of drainage in the ambulant patient, who carries out his normal duties without embarrassment. At the same time antibiotic treatment is instituted, combined with repeated antral lavage.

At the outset it is necessary to establish the nature and extent of the infection. If the ethmoids are infected then it is advisable to treat these first; for this, Proetz displacement therapy is very useful, as it is also in cases where the frontals, ethmoids, and sphenoids are also infected. The best results of the method described in this article are obtained where the infection is predominently in the antrum.

The series presented in this short paper consists of about 35 cases, but one hopes that some otologists will be stimulated to adopt this method in suitable cases and explore its possibilities further.

Technical Details

First a proof puncture with a 7·5 mm. Lichtwitz cannula is performed, and the surgeon aspirates for secretions, which are then sent to the laboratory for bacterial investigations. Now he proceeds to irrigate the antrum with warm normal saline till the return flow is clear. When this occurs a solution of freshly prepared Rondase (Evans) is introduced into the antral cavity with an ordinary glass syringe that will adapt to the cannula. After 3 minutes a freshly prepared solution of soluble Terramycin or Erythrocin or other soluble wide-field antibiotic is introduced in a similar manner. A vial of 100 mg. of Terramycin dissolved in not more than 2 c.c of sterile water or saline is usually employed.

Finally polythene tubing of 1 mm. bore is introduced into the antrum and threaded through the hollow of the cannula. It is necessary to cause it to curl up, and so leave at least 2 inches of this tubing in the antral cavity. This will allow sufficient length to be withdrawn for lavage on the next occasion. The tubing is now cut short at the vestibule of the nose and tucked into the nostril out of sight.

Antral lavage is carried out through this tube on successive occasions by means of a suitable needle and a 20 c.c. syringe. On each occasion it may be necessary to introduce Rondase and antibiotic solutions until there is definite improvement in the colour and quantity of the returned secretions. Each case should be judged on its own merits when deciding on the frequency of lavage and the use of antibiotics. When the laboratory report becomes available the appropriate antibiotic can be used. The majority of cases have responded very satisfactorily to the routine use of Terramycin. In recent infections the early response is dramatic and the return to normality early; in chronically infected cases, the response is slower but further treatment for several weeks will bring a clear return in most cases.

I believe that a sufficient amount of antibiotic finds its way into the adjacent ethmoidal cells, which fact in turn augurs well for infections of the frontal sinuses, whose impeded drainage is relieved by reduction of the inflammatory oedema in the neighbourhood of the frontonasal duct.

In cases where ethmoidal-cell infection is suspected, it is necessary to consider the use of displacement therapy immediately after antral lavage. If there is muco-pus in the return flow of the displaced fluid after lavage, then one can take it that the ethmoids are also infected. This combined therapy has found its greatest success in infections in children, who are not intolerant of the polythene and generally are most amenable to displacement therapy.

If necessary, lavage with or without instillations can be carried out for a period of 2 months after initial insertion of the tubing. Before the polythene is removed one or two periods of one week without any treatment should elapse, after which a clear return is a good enough signal to remove the tube, which glides out painlessly. The polythene turns black after one month but this is of no significance. Should the return fluid not be reasonably clear after 4 weeks, the ultimate result may be in some doubt. Perseverence for a further few weeks will decide the next step and, should the treatment fail, we have a strong case for surgery.

After three weeks, hypersensitivity of the maxilla and teeth results from pressure of the tube on the antral mucosa. Slight withdrawal generally relieves this neuralgia. The polythene tube has not been lost in a single case, but the

possibility exists. This risk can be removed by expanding the protruding tip with a hot metal instrument.

Results

X-ray examination is advisable in most cases before treatment. In several cases a repeat X-ray after treatment confirmed the clinical cure. The plates of one case showing the radiological return to normality are most impressive, after a typical history of nearly 20 years' infection. The patient a lawyer, was considered by several otologists whom he consulted over the years to be an undoubted case for surgery, especially as his antrum was completely opaque on X-ray. In all he had 5 weeks of concentrated treatment in the manner outlined above. Six months have elapsed and he still seems to be normal both clinically and radiologically. In two other cases infection and symptoms returned after a period of several weeks. Both these cases were agreeable to surgery at this stage and seem to be doing well after intranasal antrostomy.

The indwelling polythene tube has been used by otologists for some years, but it has been usual to remove it after about 5 days. By leaving it in longer, one creates a temporary antrostomy with the added advantage of concentrated topical antibiotic therapy. In cases that have had an antrostomy done before, the fluids will escape and local therapy is bound to fail. In this relatively simple form of conservative treatment the progress of the infection can be gauged very accurately. The amount of secretion and its colour and nature are always clearly discernible during the course of treatment. It is interesting to note that in favourable cases the bitter taste of the antibiotic persists for some time after its instillation, whereas in cases where the cilia are not actively sweeping this taste appears to be absent, and stale and discoloured antibiotic can be seen with the next lavage. Generally, the persistent presence of stale pale threads after 4 weeks of treatment is an unfavourable sign.

The efficacy of this form of therapy was shown in a case of severe empyema of the antrum, following a cold. The patient, a headmaster, went back to teach after 6 days in hospital, with the tube tucked away in his nasal vestibule for a further period of 10 days, and none of the boys or masters spotted it.

This treatment has appeared to be promising in a few cases of chronic postnasal drip of infective origin, and is being tried in a further series for accurate evaluation.

Children tolerate the tubes quite well. In one case of bilateral purulent infection with middle-ear deafness, they were tolerated for 18 days with remarkable improvement in the deafness. I feel that this treatment combined with Proetz displacement will reach its greatest height of success in young children with resistant infection. Such treatment could be considered as a preliminary conservative measure in almost every case where surgery is already indicated. Even in advanced cases it would be an excellent cleansing measure. It was used in all cases where proof puncture was usually indicated, even in those where one thought a single puncture would suffice. It was revealing to see the amount of pus secreted in these cases after the initial puncture. It makes one think how often a single puncture may have been insufficient and the patient sent away with smouldering infection. Considering how unfavourably the ostium is placed for gravitational drainage, it is just as well that the power of recovery of the antral mucosa is so efficient.

SUMMARY

A method of treatment of acute and chronic infections in the paranasal sinuses is described. This treatment is particularly applicable in infections in children. The introduction of polythene plastic tubing into the antrum through a Lichtwitz cannula is first necessary. The antrum is then repeatedly washed out, followed by the introduction of a diffusing agent like Rondase and then by a wide-field antibiotic. This process is repeated till the returned fluid from the antral cavity is clear, even if the tube has to be retained for a period of 4-8 weeks.

Proetz displacement therapy may be necessary in conjunction with this treatment if infection of the ethmoids coexists. Observations of the character of the antral fluids is most revealing as regards prognosis and choice of further treatment. A stronger case for surgery can be deducted from this procedure in cases that have failed to respond favourably.

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EDITORIAL

NUCLEAR EXPLOSIONS

Pleas for banning the testing of nuclear weapons are kept much in the public eye these days, and there will be a good deal of sympathy for the inhabitants of the Japanese archipelago, which is uncomfortably placed between the Scylla of the Russian testing grounds and the Charybdis of the Anglo-American ones. It is ironic that the only people to have experienced the fury of the real thing should now be exposed to the more insidious fall-out of the H-bomb trials. At Easter-tide, with its goodwill messages, the Japanese Government issued warning to its people that radio-active rain was falling and that they should wash all vegetables to try and rid them of the noxious contamination from the skies.

The atomic age has created a new branch of medicine that is bound to influence enormously both research and practice. We have recently referred to the value and usefulness of radio-active isotopes.[1] The other side of the balance sheet tells a sombre tale, awful in its implications, boding no good for the human race.

Whenever an explosion takes place—or, for that matter, whenever nuclear power is used in a 'peaceful' reactor—radio-active isotopes, over 200 in number, are set free.[2] All are potentially harmful to man but the extent to which the worst of them are generated depends upon the circumstances of the explosion. For instance, the waste from a modern atomic reactor is radio-active, so that it cannot be merely poured down the drain—special precautions have to be taken in disposing of it. But this is a problem that can be rationally solved by scientists when the time comes. When a weapon is exploded, the isotope fall-out is controlled by studying prevailing winds and also by controlling the height of the explosion. If the bomb is detonated on or near the surface of the earth—e.g. the 'old-fashioned' (!) atomic bomb exploded at Hiroshima—the fall-out is local in nature, extending radially beyond the confines of the blast damage. At Hiroshima all people within a half-mile radius of the site of explosion were killed, and beyond that perimeter about half died within a radius of 2 miles. But most of these deaths were due to the blast; the radio-active fall-out casualties were relatively few. With an explosion on the ground and a favourable wind, however, this deadly

VAN DIE REDAKSIE

KERNONTPLOFFINGS

Pleidooie om die toets van kernwapens te verban, word tans gedurig onder die aandag van die publiek gebring, en daar sal heelwat simpatie wees vir die inwoners van die Japannese eilandgroep wat ongerieflik tussen die Scylla van die Russiese toetsvelde en die Charybdis van die Anglo-Amerikaanse velde geleë is. Dit is ironies dat die enigste mense wat die woede van die ware ding ondervind het, nou aan die gevaarliker neerslag van die waterstofbom-proefnemings blootgestel sal word. Gedurende Paastyd, met sy welwillendheidsboodskappe, het die Japannese Regering sy mense gewaarsku dat radio-aktiewe reën besig was om te val en dat hulle alle groente behoort te was in 'n poging om van die verraderlike besoedeling uit die lug ontslae te raak.

Die atoom-eeu het 'n nuwe tak van geneeskunde daargestel, wat stellig beide navorsing en praktyk geweldig sal beïnvloed. Ons het onlangs verwys na die waarde en bruikbaarheid van radio-aktiewe isotope.[1] Die ander kant van die balansstaat vertel egter 'n somber verhaal met skrikwekkende implikasies, wat niks goeds vir die mensdom inhou nie.

Elke keer wanneer 'n ontploffing geskied—of, inderdaad, elke keer wanneer kernkrag in 'n ‚vreedsame' reaktor gebruik word—word radio-aktiewe isotope, meer as 200 in getal, vrygestel.[2] Almal is potensieel skadelik vir die mens, maar die mate waar tot die skadelikste van hulle opgewek word, hang van die omstandighede van die ontploffing af. Byvoorbeeld, die afvalmateriaal van 'n moderne atoomreaktor is radio-aktief, sodat dit nie sommer net weggegooi kan word nie—spesiale voorsorgsmaatreëls moet getref word om daarvan ontslae te raak. Maar dit is 'n probleem wat wetenskaplikes op 'n verstandige wyse kan oplos wanneer dit tyd daarvoor is. Wanneer 'n wapen ontplof word, word die isotoopneerslag beheer deur bestudering van heersende winde, en ook deur die hoogte van die ontploffing te beheer. As die bom op of naby die oppervlakte van die aarde ontplof word—bv. die ‚outydse' (!) atoombom wat by Hiroshima ontplof is—is die neerslag plaaslik van aard en strek radiaal tot anderkant die omvang van die ontploffingskade uit. By Hiroshima is alle mense binne 'n omtrek van 'n halfmyl vanaf die plek van ontploffing gedood en anderkant daardie perimeter het ongeveer die helfte binne 'n omtrek van 2 myl gesterf. Maar die meerderheid van hierdie sterfgevalle was aan die ontploffing te wyte; die ongevalle weens die radio-aktiewe neerslag was betreklik gering. Met 'n ontploffing op die grond en 'n gunstige wind, kan hierdie dodende wolk egter oor honderde myle sprei. Dit is inderdaad verklaar dat, deur 'n waterstofbom in die noorde van

cloud might be carried over many hundreds of miles. In fact, it has been said that by dropping an H-bomb in the north of England when suitable winds prevailed, the entire population of the British Isles might be wiped out by the fall-out.

The hazard to human health from the nuclear testing that is being carried out at the present time arises from explosion of the bomb at high altitude. This method eliminates local fall-out of radio-active elements but projects them upwards instead, where they remain stored in the 'stratosphere reservoir'. Their subsequent fate depends upon their individual properties. Some highly damaging ones like strontium-89 and iodine-131 are too short-lived, biologically speaking, to survive storage for long periods. Some possess long lives, however, and strontium-90, which is the main source of radio-active contamination from the tests, is good for about 15 years of activity. Strontium-90 descends very slowly (like the gentle rain from heaven), at about 10% a year, and this allows it to spread all over the globe, before settling on the vegetation and soil.

The radiations given off by strontium-90 are readily absorbed by the walls of houses and by clothing and enter the body when contaminated greenstuff and fruit are eaten or the milk from contaminated animals is drunk. Strontium-90 is chemically similar to calcium and is therefore taken up and concentrated by the bones, where it may remain radio-active for from 5 to 10 years. It is concentrated particularly easily in the bones of growing children and, according to British and American official reports,[3,4] some children in both countries have already accumulated measurable amounts in their bodies. Strontium-90 is deposited by predilection in the vertebrae and sternum. Children under 4 years of age are said to take up to 5 times as much as an adult.

The main hazard is a somatic one, since strontium-90 emits β-rays which do not penetrate to the gonads. (This, however, does not mean that genetic disturbances may not result from nuclear warfare.) The somatic hazards are bone lesions such as sarcomata and the leukaemias, caused by the deposition in the skeleton of the comparatively long-lived strontium-90. In this respect it behaves like radium, which is known to produce sarcomata in bone after long exposure. Mutations can be caused by ionizing radiation, and some naturally-occurring mutations in man result from cosmic rays and rays from radio-active rocks and radio-active materials present in the human body. One of the effects of the test explosions is to increase the level of background radiation; this may produce additional genetic mutations, the majority of which are harmful.

Only the surface of the problem has so far been exposed, and as yet almost none of the questions that spring to mind can be answered. No one knows the maximum tolerance concentration of strontium-90 for human bones: British scientists believe that the 'body burden' of North Americans will have reached the critical level by the early 1970s, but the Americans themselves disagree, being less pessimistic over the present uptake in human bones. No one can tell Engeland te los wanneer die wind geskik is, die hele bevolking van die Britse Eilande deur die neerslag uitgewis kan word.

Die gevaar wat die kernproefnemings, wat tans gedoen word, vir menslike gesondheid inhou, ontstaan deur ontploffing van die bom op 'n hoë hoogte. Hierdie metode skakel plaaslike neerslag van radio-aktiewe elemente uit, maar in plaas daarvan, word hulle boontoe opgeskiet, waar hulle in die ‚stratoferiese bergplek' opgegaar bly. Wat daarna met hulle gebeur, hang van hulle individuele hoedanighede af. Sommige hoog-beskadigende soorte, soos bv. stronsium-89 en jodium-131 se lewensduur is, biologies gesproke, te kort om lang periodes van opgaring te oorleef. Sommige se lewensduur is egter langer en stronsium-90, wat die hoofbron van radio-aktiewe besoedeling as gevolg van die toetse is, se aktiwiteit duur ongeveer 15 jaar. Stronsium-90 daal baie stadig na benede (soos die sagte reën uit die hemel), teen ongeveer 10% per jaar, en dit bring mee dat dit oor die hele aardbol kan sprei voordat dit op die plantegroei en grond te lande kom.

Die strale wat deur stronsium-90 uitgestraal word, word maklik deur die mure van huise en klere geabsorbeer, en gaan die liggaam binne wanneer besoedelde groente en vrugte geëet, of melk van besoedelde diere gedrink word. Chemies stem stronsium-90 en kalsium ooreen, en word dus deur die bene in die liggaam opgeneem en gekonsentreer, waar dit vir 'n tydperk van 5-10 jaar radio-aktief kan bly. Dit word veral maklik in die beenstelsels van groeiende kinders gekonsentreer en, volgens die Britse en Amerikaanse offisiële verslae,[3,4] het sommige kinders in beide lande reeds meetbare hoeveelhede daarvan in hulle liggame gestoor. Stronsium-90 word by voorkeur in die werwels en borsbeen neergeslaan. Dit word verklaar dat kinders onder 4 jaar 5-maal soveel daarvan as 'n grootmens absorbeer.

Die grootste gevaar is somaties van aard, aangesien stronsium-90 β-strale afgee wat nie tot die geslagskliere deurdring nie. (Dit beteken egter nie dat genetiese steurings nie as gevolg van kernoorloë sal ontstaan nie.) Die somatiese gevare is letsels aan die beenstelsel soos bv. sarkome en die verskillende soorte leukemie, wat deur die neerslag in die skelet van die betreklik lank-lewende stronsium-90 veroorsaak word. Hierin stem dit met radium ooreen, wat bekend is dat dit na lang blootstelling sarkome in die beenstelsel veroorsaak. Mutasie kan deur ioniserende bestraling veroorsaak word en sommige natuurlik-voorkomende mutasies by die mens word deur kosmiese strale en strale van radio-aktiewe klippe, en radio-aktiewe stowwe, wat in die menslike liggaam aanwesig is, veroorsaak. Een van die uitwerkings van die toetsontploffings is om die vlak van agtergrondbestraling te vermeerder; dit mag bykomende genetiese mutasies voortbring, die meerderheid waarvan skadelik is.

Slegs die oppervlakte van die probleem is tot dusver ontbloot en tot nog toe kan nie een van die vrae wat by 'n mens opkom, beantwoord word nie. Niemand weet wat die maksium verdraagsaamheidskonsentrasie van stronsium-90 vir die menslike beenstelsel is nie: Britse wetenskaplikes glo dat die ‚liggaamslas' van Noord-Amerikaners die kritiese vlak kort na 1970 sal bereik, maar die Amerikaners self stem nie hiermee ooreen nie, aangesien hulle nie so pessimisties is oor die huidige opname in menslike beenstelsels nie. Niemand kan sê hoe lank dit sal duur

the interval of time that will elapse before the first strontium-induced lesions appear. In fact, all we know for certain at present is that, as long as nuclear explosions continue in the world, a potentially-lethal store of strontium-90 is being built up in the bones of every member of the human family.

1. Editorial (1957): S. Afr. Med. J., **31**, 529.
2. Editorial (1957): Brit. Med. J., **1**, 752.
3. *The Hazards to Man of Nuclear and Allied Radiation* (1956): U.K. Med. Res. Coun. London: H.M. Stationery Office.
4. *Unmeasured Hazards* (1956): London: World Federation of Scientific Workers.

voordat die eerste letsels wat deur stronsium veroorsaak is, te voorskyn sal kom nie. Inderdaad, al waaroor ons tans sekerheid het is dat, solank daar kernontploffings in die wêreld geskied, 'n potensieel-dodende voorraad van stronsium-90 in die beenstelsels van elke lid van die menslike geslag opgebou word.

1. Van die Redaksie (1957): S. Afr. T. Geneesk., **31**, 529.
2. Van die Redaksie (1957): Brit. Med. J., **1**, 752.
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TRISMUS IN CHILDREN

WITH SPECIAL REFERENCE TO POST-VACCINAL ENCEPHALITIS

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Trismus is a rare finding in children. By trismus is meant a *maintained* closure of the jaws by muscular spasm so that the mouth cannot be opened. Clenching of the jaws occurs as a convulsive manifestation, and mumps, alveolar abscess or other local lesions can cause a mechanical inability to open the mouth, but these phenomena do not constitute true trismus.

The commonest cause of this sign in children is tetanus, seen fairly frequently at Baragwanath Hospital, where approximately 12 cases of tetanus neonatorum occur among 5,000 annual paediatric admissions. Trismus as a feature in other diseases has not previously been encountered in the paediatric unit, and it is for this reason that the following 4 cases are presented, together with a 5th case from Edenvale Hospital, Johannesburg.

Case 1 is of special importance because, as far as we are aware, it is the first reported case of post-vaccinal encephalitis proved pathologically in South Africa.

CASE 1

A female child aged 2 years 5 months was admitted on 28 August 1955 with a history of peculiar behaviour for 1 day. She had been quite well until 2 weeks previously, when she was vaccinated for the first time. Since then the child had complained of a painful arm and had been slightly unwell. On the day before admission she behaved queerly, not responding when spoken to, and refusing feeds. She had received one injection and 3 tablets of unknown substances from a private doctor. On the day of admission the father noticed that the child was having difficulty in opening the mouth.

Examination. The patient was a well nourished child weighing 30 lb. Her temperature was 102 F, pulse rate 126 per minute, and respiration rate 24 per minute. She was stuporose and mildly dehydrated. Her respiration was deep and suggested the possibility of acidosis. On the left arm there was a scabbed vaccination mark, and on the left leg a similar lesion was noted. Trismus was present, and the throat could not be examined. There were rhonchi throughout both lung fields. No neck stiffness and no Kernig's or Brudzinski's sign were found. There were no obvious cranial-nerve palsies, all reflexes were present, and plantar reflexes were flexor. The fundi were normal. Twelve hours later the mouth could be opened sufficiently to observe that both tonsils were enlarged and infected; the right pupil was then smaller than the left, and the child appeared to be more stuporose. Two days after admission the pupils were equal in size, the right knee jerk was not obtained, but other reflexes were present and the plantar reflexes were normal. The child had become deeply comatose, and had remained pyrexial since admission. She died early on the 4th hospital day soon after becoming hyperpyrexial.

Investigations. The cerebrospinal fluid showed 4 polymorphonuclear leucocytes and 19 lymphocytes per c.mm., protein 27 mg.%, sugar 45 mg.%, and chloride 730 mg.%. A leucocytosis (14,300 leucocytes per c.mm.) and a slight acidosis (plasma carbon-dioxide combining power 18·6 mEq per litre), were present. Blood smears, urine, blood urea and serum-salicylate levels were normal.

Treatment. Intravenous dextrose and saline solutions were given for 24 hours, and thereafter full-strength cow's-milk feeds by naso-gastric tube. Parenteral penicillin was administered 6-hourly.

Pathology

At autopsy recent vaccination scabs were observed on the left arm and left leg, each about 1 cm. in diameter. The brain was congested but showed no other obvious abnormality.

Sections were taken from various viscera, but showed no relevant features.

Sections prepared from various parts of the brain showed lesions in almost all of the sections taken, but these varied in severity. The lesions had a uniform character throughout and took the form of perivascular infiltrations of non-granular cells, mainly lymphocytes, although monocytes were also prominent (Fig. 1). The white matter surrounding the vessels showed extensive degenerative changes, with an accumulation of cells consisting mainly of microglial phagocytes and other reactive cell types (Fig. 2); Sharlach R. preparations showed that many of these contained fatty material. The extent of this process in the white matter varied according to the site of the lesions, but faded off gradually into the surrounding brain tissue. Myelin preparations showed definite myelin degeneration in the perivascular regions at the site of the lesions (Fig. 3), although at this stage of the disease process the margin of myelin loss was not clear-cut. Nerve cells in the vicinity of the lesions showed chromatolysis. The

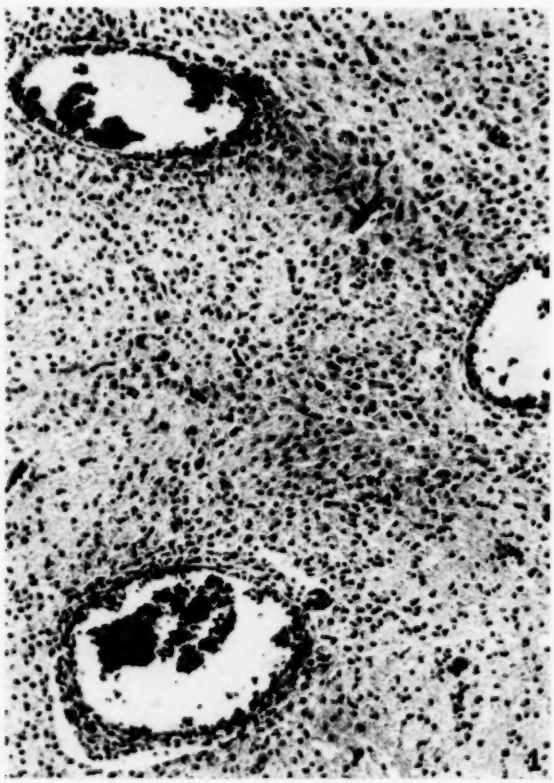

Fig. 1. Case 1. Perivascular lymphocytic infiltration and reactive changes in the surrounding tissue. H and E × 120.

leptomeninges showed a slight to moderate infiltration by non-granular cells, mainly lymphocytes.

The lesions were distributed maximally in the midbrain, pons and medulla. In the midbrain the focal lesions reached great intensity near the mid-line, in the region of the peri-aqueductal nuclei and the substantia nigra. They were of considerable intensity throughout the entire structure of the pons. The medulla oblongata showed focal lesions throughout, including the region of the inferior olivary nuclei. In the white matter of the cerebral hemispheres the lesions were not generally so extensive as those in the brain stem or midbrain, but were fairly widespread in the subcortical and paraventricular white matter. In the basal ganglia, thalamus and hypothalamus the lesions were of relatively minor degree. The cerebellum showed isolated focal lesions of moderate intensity in the white matter of the hemispheres, but the vermis was largely spared. The spinal cord was not available for examination.

These findings are consistent with the diagnosis of *post infective encephalitis* following vaccination.

CASE 2

A male infant aged 20 months was admitted on 12 March 1956 with a history of convulsions and mild diarrhoea for 2 days. Previously quite well, he had had 2 convulsions on each of the 2 days preceding admission. The convulsions resembled major epileptic seizures. On the day of admission he had a convulsion at 6 a.m. and became comatose.

Examination. The patient was a well nourished child weighing 19 lb., and was not dehydrated. His temperature was 103°F, pulse rate 124 per minute, and respiration rate 28 per minute. He was comatose. Trismus was present and his throat could not be examined. Muscle tone was poor and there was no neck rigidity, nor could Kernig's or Brudzinski's sign be elicited. All reflexes were present and equal, and plantar reflexes were flexor. His pupils were equal and reacted to light, and his discs were normal. Apart from the trismus there did not appear to be any abnormalities of the cranial nerves. He presented therefore as an unconscious hypotonic child with trismus and pyrexia, a history of convulsions and gastro-enteritis. Intravenous glucose had no effect on the coma, and 10 hours after admission he became hyperpyrexial, with a temperature of 106°F. He collapsed and died 18 hours after admission.

Investigations. The cerebrospinal fluid showed 130 polymorphonuclear leucocytes and 95 lymphocytes per c.mm., protein 33 mg.%, sugar 55 mg.%, and chloride 810 mg.%. Microscopic examination showed no organisms, and cultivation yielded no growth. A mild hypoglycaemia (blood sugar 42 mg.%) and a leucocytosis (19,800 leucocytes per c.mm.) were present. The urine showed a trace of sugar and acetone. The blood urea was 59 mg.%. Blood and rectal swab cultures were negative.

Treatment. An injection of 10 c.c. of 50% glucose was given intravenously, and milk feeds were given by naso-gastric tube. Chloromycetin was administered 6 hourly.

Pathology

Macroscopic findings in this case at autopsy were again non-contributory, apart from marked congestion of the central nervous system. The significant microscopic findings were essentially those of *acute anterior poliomyelitis* affecting the spinal cord (Fig. 4), and of *polio-encephalitis* in the brain stem.

As is characteristic of this infection the nerve cells related to motor nuclei were severely damaged, particularly in the spinal

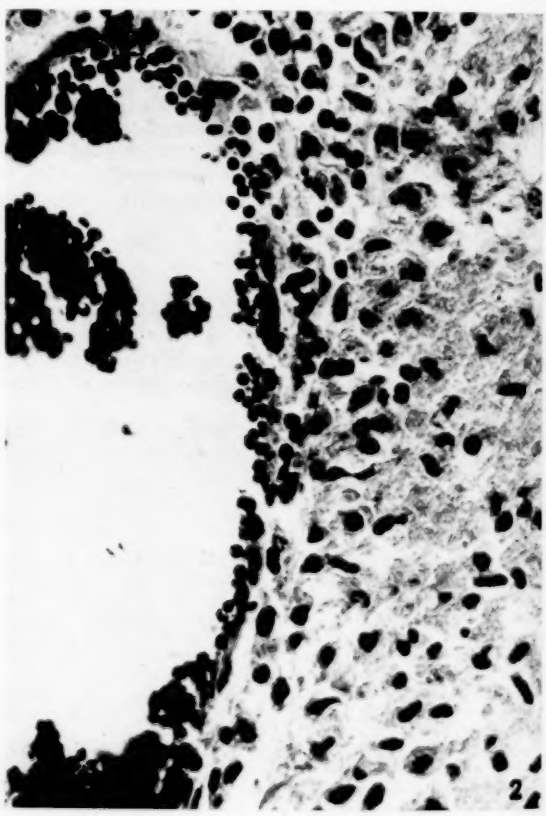

Fig. 2. Case 1. High-power view of microglial phagocytic proliferation in degenerating perivascular tissue. H and E × 480.

cord, and inflammatory responses were centred largely in the region of these nuclei (Fig. 5). Perivascular lymphocytic cuffing was marked but there was no evidence of myelin degeneration.

The distribution of the lesions is of some importance. The spinal cord was involved maximally in the lumbar region. The medulla oblongata showed scattered lesions, particularly in the nuclei in the floor of the 4th ventricle. The inflammatory lesions in the pons were confined to the tegmentum. The leptomeninges were infiltrated by lymphocytes.

CASE 3

A female infant aged 3½ months was admitted on 3 May 1956 with a history of cough for 2 days, some diarrhoea at the onset of the cough, fever, and rapid respiration for 1 day. The mother thought there was increased fullness of the fontanelle.

Examination. The patient was a well nourished baby weighing 13 lb., very ill indeed, and extremely dyspnoeic, with grunting rapid respiration and costal recession. The rectal temperature was 101°F, pulse rate 158 per minute, and respiration rate 48 per minute. The child was extremely hypotonic. Trismus prevented examination of the mouth and throat. The child appeared to be conscious. Neither Kernig's nor Brudzinski's sign was present and there was no neck rigidity. The fontanelle was slightly full, but neither tense nor bulging.

Investigations. Lumbar puncture revealed a purulent cerebrospinal fluid. Numerous pus cells, but no organisms, were seen microscopically, and cultivation yielded no growth. The protein content was greater than 200 mg.%. There was insufficient fluid for sugar and chloride estimation.

Treatment. High-dosage penicillin, streptomycin and sulphatriad therapy was begun, but the infant died 8 hours after admission.

Permission for post-mortem examination could not be obtained.

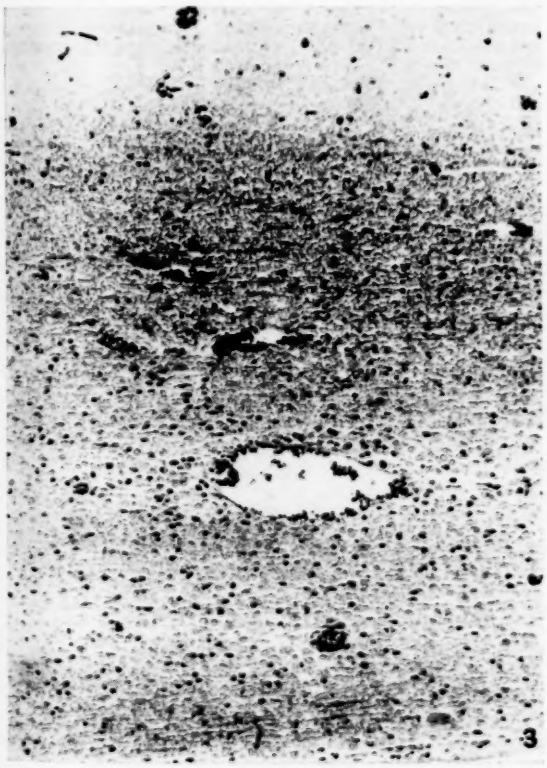

Fig. 3. Case 1. Perivascular myelin degeneration in subcortical white matter. Weil's method for myelin × 120.

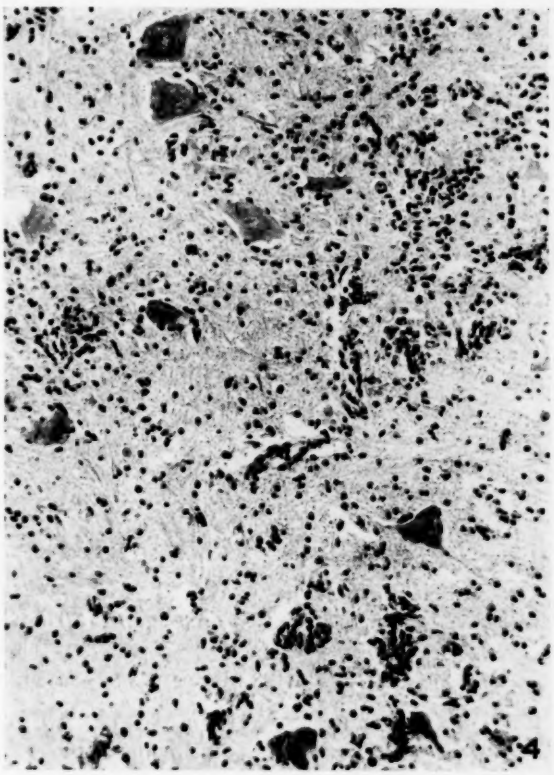

Fig. 4. Case 2. Typical lesions of acute anterior poliomyelitis, with focal inflammatory reaction, chromatolysis and destruction of motor nerve-cells. Cresyl violet × 120.

CASE 4

A female child aged 1 year 9 months was admitted on 1 June 1956 with a history of convulsions for the previous 3 hours. The convulsions had been seen chiefly on the right side, and the child had appeared feverish since the onset of fits. There had been no cough, diarrhoea or previous fits.

Examination and Course

The patient was a well nourished child weighing 20 lb. She was having frequent generalized convulsions. The temperature was 102°F, pulse rate 120 per minute, and respiration rate 40 per minute. All reflexes were present and equal and the plantar responses were flexor. No neck rigidity and no Kernig's or Brudzinski's sign were found. There was a doubtful increase of tone in the left arm and leg. Rhonchi and crepitations were present throughout both lung fields.

By next day the abnormal signs in the chest had disappeared. Generalized convulsions had ceased, but twitchings of the left side of the face and the left hand were present. The child was extremely hypotonic, and the only reflex that could be obtained was the left biceps jerk. She never became conscious, but became more comatose each day. On the 3rd day of her illness she stopped having fits. With the exception of the ankle jerks all reflexes were now obtainable. She was still extremely hypotonic, and a divergent squint was observed. Trismus was now present, and prevented examination of the mouth and throat. On the 4th day, however, the mouth could be opened just sufficiently to see the pharynx. Knee and ankle jerks were lost, and a weak ankle clonus had appeared on the left side. The blood pressure was 80/20 mm. Hg. There was doubtful blurring of the nasal margin of the optic discs.

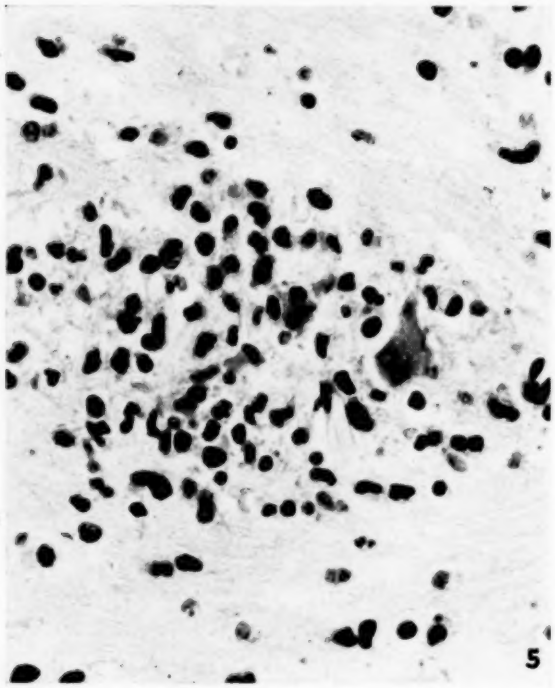

Fig. 5. Case 2. High power view of a polio-encephalitic lesion in the brainstem, with inflammatory and glial-cell reaction, associated with nerve-cell degeneration. Cresyl violet × 480.

The child was pyrexial throughout. Her stools, although loose and mucoid, were small and never frequent. She did not vomit or cough.

She died on the 5th day.

Investigations

On 1 June the cerebrospinal fluid showed 6 polymorphonuclear leucocytes, 4 lymphocytes and 3 erythrocytes per c.mm., protein 17 mg.% sugar 113 mg.% and chloride 615 mg.%. On 4 June the cell count had increased to 73 polymorphonuclear leucocytes and 5 lymphocytes per c.mm. and protein had increased to 31 mg.%; sugar 65 mg.% and chloride 625 mg.%.

X-ray of chest on 4 June showed a patch of opacity confined to the apex of the dorsal segment of the right lower lobe.

There was a polymorphonuclear leucocytosis (leucocytes 16,200 per c.mm.—75% polymorphonuclear leucocytes, 7% monocytes, 18% lymphocytes).

On 4 June a mild acidosis (carbon dioxide combining power 18 mEq/litre) and marked hyponatraemia (serum sodium 120 mEq/litre) were present.

Serum potassium, blood urea, blood sugar, haemoglobin and red cells were normal.

An intradermal injection of 0·00002 mg PPD was negative.

Neither polio virus nor coxsackie virus was isolated from the stools.

Treatment

Penicillin G by injection and sulphonamide were given for 3 days, followed by penicillin V orally; intramuscular phenobarbitone was used for control of the convulsions.

Three pints of sweetened full-strength cow's milk were given by naso-gastric tube in divided feeds daily.

Pathology

At autopsy 3 small localized focal necrotic lesions were seen in the liver, which was otherwise normal in appearance. The brain appeared swollen, with a suggestion of uncal and cerebellar pressure cones. Sections taken from various viscera showed nothing of significance apart from the localized and encapsulated necrotic lesions in the liver. These might possibly have been tuberculous in origin. No etiological factor however was observed in specially prepared preparations.

Numerous sections from the brain showed minimal evidence of perivascular or parenchymatous inflammatory-cell infiltration. In many areas, however, including the cerebral cortex, the nerve cells showed well-marked eosinophilic shrinkage indicative of ischaemic atrophy. 'Encrustations' suggesting anoxia were also prominent, particularly in the hypothalamus. In the region of the mid-pontine tegmentum, an occasional isolated focus of microglial aggregation suggesting neuronophagia was found (Fig. 6). An occasional small vessel was surrounded by a cuff of lymphocytes. No other lesion of this type could be detected in any of the other brain sections. Myelin preparations showed an essentially normal picture. There was no histological evidence of a meningeal reaction.

The histological features therefore are in favour of encephalopathy with severe ischaemia and anoxia rather than a virus encephalitis. These changes may, of course, be due to circulatory disturbances occurring in association with convulsions. The isolated focal lesion in the mid-pontine tegmentum with a local cell reaction may have been responsible for the trismus.

CASE 5

A female child aged 6 years was admitted to Edenvale Hospital on 24 November 1956. One week earlier she had had a slight cough and abdominal pain, but had been attending school for the previous week. On the night before admission she refused her supper, and at 5 a.m. on the day of admission she had a generalized convulsion and became unconscious.

Examination. The patient was a well nourished comatose child. Her temperature was 96·4°F, pulse rate 100 per minute, and respiration rate 28 per minute. The child was hypotonic with absent reflexes. No neck stiffness and no Kernig's or Brudzinski's sign were found. Trismus prevented examination of the mouth and throat. She had a left-sided, and then a generalized, convulsion

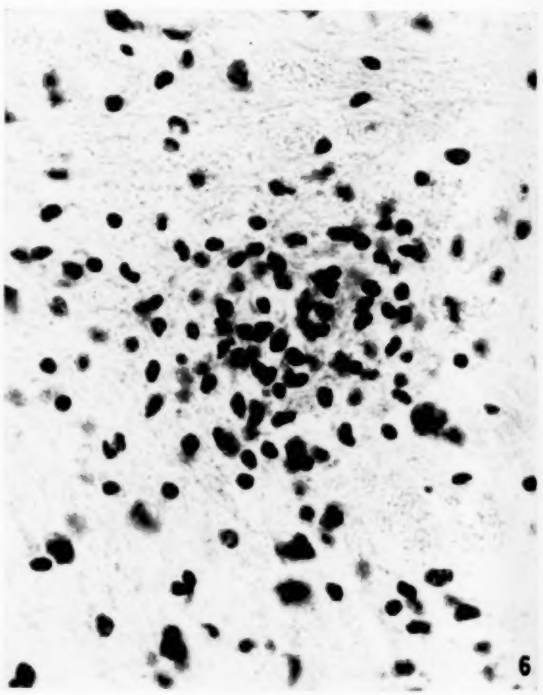

Fig. 6. Case 4. Isolated microglial aggregation in the mid-pontine tegmentum. Cresyl violet × 480.

soon after receiving 50% glucose intravenously. The glucose had no effect on the coma. She died 7 hours after admission, without becoming conscious.

Investigations. The cerebrospinal fluid showed no cells, protein 12 mg.%, chloride 740 mg.%, and sugar 12 mg.%. A severe hypoglycaemia was present, with the blood sugar level 14 mg.%. There was a marked leucocytosis (126,000 per c.mm.). The differential count showed a lymphocytosis and an absolute neutrophilia (23·5% polymorphonuclear leucocytes, 5·5% monocytes, 63·5% lymphocytes, 2·5% eosinophils, 1·0% basophils). There were 1·0% late normoblasts present. The urine passed after intravenous sugar solutions contained no albumin but sugar +++.

Treatment. 500,000 units of penicillin were given intramuscularly, and 50 c.c. of 50% glucose intravenously, followed by a slow intravenous drip of 10% invert sugar in water.

Pathology

Apart from congestion of the brain, no obvious abnormalities were detected at autopsy.

Sections of various viscera were examined but showed no relevant changes. Blocks from the liver, suprarenal and pancreas in particular were normal in appearance.

The specimen of brain tissue submitted for histological examination consisted of part of the brain stem, basal ganglia, and part of the frontal and temporal lobes.

Numerous representative sections taken from the specimen of brain tissue, including the pons, showed similar changes throughout. Intense congestion of the small blood vessels was most striking in all sections, and this was accompanied by perivascular haemorrhage in many areas. Many of the larger neurones showed evidence of chromatolytic change, and eosinophilic ischaemic atrophy was obvious in certain areas, particularly in the cornu ammonis. There was no evidence of a parenchymatous inflammatory lesion, nor was there a significant degree of perivascular lymphocytic cuffing present. The white matter showed changes indicative of cerebral oedema. In the lumen of some of the larger blood vessels considerable aggregations of lymphocytes could be seen. Preparations stained by Weil's method showed no evidence of demyelination. The histological features suggested an *encephalopathy in association with whooping cough.* No evidence of the features of a virus encephalitis was detected in any of the sections examined.

DISCUSSION

The exact site of the neurological lesion underlying trismus is uncertain, but involvement of the 5th cranial nerve, which supplies the masseter, or of its motor nucleus is likely. Matzke and Baker's study in poliomyelitis[1] supports this. Although the lesions in the cases described were widespread, the mid-pontine tegmentum (the region of the 5th-nerve motor nucleus)[2] was involved in all the cases described above which were examined histologically.

Case 1 is an example of post-vaccinal encephalitis, and in this condition, and the encephalitis following variola, Ford[3] states that trismus is a common sign, and may be so prominent as to suggest tetanus. Other evidence of bulbar involvement has been reported in this encephalitis, as for example the palsy of the abducens and oculo-motor nerves which is sometimes observed.

In case 1 extensive lesions were demonstrated throughout the pons, including the vicinity of the mid-tegmental nuclei. The pathology in this brain is consistent with that described in various reports[4-8] of post-infective encephalitis, where the majority of authorities agree that the process is largely one of perivascular, or more precisely perivenous, myelin destruction, accompanied by a cellular response. The lesions are of the same character as those found in experimental acute encephalomyelitis, where the changes are a response to inoculated cerebral tissue, and indicate a hypersensitivity reaction.[9-11] The pathological appearances of the brain vary according to the duration of illness. Initially there is naturally a considerable cellular reaction with myelin destruction, but a clear-cut margin to the demyelinated zone may only be obvious when the active phase is subsiding.[7] Free fat may be seen in microglial phagocytes during active myelin destruction. In this case the process is in a relatively early phase.

It is interesting to compare the character of the lesions of a true virus encephalitis as seen in case 2 with those in this case of post-vaccinal encephalitis. In virus infection the lesions are typically polioclastic, with primary nerve-cell damage and inflammatory reactions tending to be centred around damaged nerve cells. Post-infective lesions are apparently primarily myelinoclastic, and this effect is maximal perivascularly. The fundamental difference in the type of lesion present is strong evidence in favour of the contention that post-infective encephalitis is not due to direct invasion by the virus, but represents a hypersensitivity reaction.[4, 9]

We have found only 2 previously reported cases of post-vaccinal encephalitis in South Africa and both these patients recovered.[12] The incidence of this disease varies widely in different reports from different countries; Scott[13] states that it is about 1 : 100,000 cases of vaccination. In New York during the mass vaccinating in 1946 the incidence was 1 : 170,000. It is much rarer after revaccination, although it can occur, and is very rare with primary vaccination in infants under 1 year. The mortality rate of the disease is quoted as being 50%. In spite of this incidence elsewhere, it would seem that in South Africa, where vaccination is compulsory, the condition is almost unknown.

Case 2 is an example of polio-encephalitis with bulbar involvement affecting the 5th nerve early and predominantly. In bulbar poliomyelitis trismus has been described as one of the most interesting and important clinical disturbances of the 5th cranial nerve.[1] It appears suddenly, usually at the end of the first week of the illness, and persists from 5 to 7 days; it may be extremely painful, and eating or even talking may be impossible. Adequate removal of secretions may be difficult, and the resulting airway obstruction may necessitate urgent tracheotomy. In some patients who recover, the involvement of the muscles of mastication is not detected until months have elapsed, when difficulty in chewing or actual atrophy of the muscles becomes apparent. The atrophy is occasionally accompanied by fibrosis which limits opening of the mouth.

Matzke and Baker[1] made detailed histological studies of the pons in 109 cases of bulbar poliomyelitis. It appears that the pons is involved in every case. The inflammatory changes appear to be most intense in the lower and upper portions of the pons, and tend to decrease greatly in the middle pontine regions. This mesodermal-glial reaction does not seem to parallel the neuronal damage, which is fairly consistent and uniform at all levels. This is particularly true of the motor nucleus of the 5th cranial nerve, which is rather severely damaged, even though inflammatory changes in its vicinity are fairly mild.

In this case, apart from the trismus, the convulsions and coma dominated the clinical picture. It is interesting in that polio-encephalitis is rare, and is said to occur in only about 2% of all cases of poliomyelitis.[14] Headache is said to be the cardinal symptom, but at the height of the illness there may be confusion, convulsions or even coma. Fanconi *et al.*,[15] in 1944, reported 375 cases of poliomyelitis with

37 cases showing cerebral symptoms, in which involvement of the sensorium occurred in 27 and 9 patients had convulsions, and the mortality rate was 10%. In 1946 Denys and Dereymaiker[16] reported 6 encephalitic cases among 23 with poliomyelitis. Two patients had convulsions, and there were alterations in the sensorium of 5. In Johannesburg in 1956 3 cases of encephalitis were seen among 99 cases of poliomyelitis.[17] This incidence conforms with experience elsewhere. In case 2 the cerebral symptoms were so striking and sudden that infection with the polio virus was not considered in the differential diagnosis made clinically.

Case 3 is an example of trismus occurring in meningitis. We have found no description of trismus in meningitis, but French [18] mentions it as occurring exceptionally in the course of severe general toxaemia as in typhoid fever, cholera or septicaemia. He says that the nature of the illness will have been diagnosed before the trismus sets in, and the symptom is quite an adventitious phenomenon. In case 3 it was this phenomenon only that led to lumbar puncture and diagnosis.

The diagnosis of *case* 4 remains obscure. Interestingly enough, a focal reactive lesion was found in the mid-pontine tegmentum, and this may have played a part in the production of the trismus. Although polio-encephalitis was the diagnosis favoured clinically, rabies was considered later in view of a convulsive disorder with muscle spasm followed by coma and death. Neurological signs and trismus may occur in the acute infantile form of Gaucher's disease,[19] but no definite evidence of any of these conditions was found on pathological examination. In view of the presence of the focal lesion in the pons with an occasional perivascular cuff of lymphocytes, a virus infection cannot be entirely excluded.

An encephalopathy of toxic origin was not considered clinically, as the cerebrospinal fluid showed 73 polymorphonuclear leucocytes per c.mm. Such an encephalopathy sometimes occurs in association with pneumonia, which was diagnosed on the clinical and radiological findings but was not seen at autopsy—the patient, however, had received treatment.

Whooping-cough encephalopathy, which may present a similar clinical picture, is not considered likely, as there had been no cough, and the leucocytosis present was predominantly polymorphonuclear in type, with only 18% of lymphocytes. This should be compared with the finding in case 5.

In view of the non-specific nature of the pathological findings the possibility of the immediate cause of death being biochemical must be considered. The biochemical investigations carried out on the 4th day of the illness, were mainly made to exclude the possibility that the flaccidity present might be due to hypopotassaemia. There was no abdominal distension and, as was expected, the serum potassium was normal.

There was no diarrhoea or polyuria, and the finding of a low serum sodium was unexpected. This child's clinical condition did not suggest sodium depletion. There was no dehydration or acidosis, which have been described in disease of the central nervous system,[20] but hyponatraemia may also be present with few apparent symptoms referable to the low blood sodium, as is commonly seen in tuberculous meningitis.

Nyhan and Cooke[20] have recently described a clinical type of hyponatraemia in disease of the central nervous system, where the symptoms are those of water intoxication, and where the convulsions respond promptly and dramatically to intravenous administration of hypertonic saline together with potassium. They suggest that the hyponatraemia is due to acute expansion of the volume of extracellular fluid in association with antidiuresis. The serum-sodium levels were very low in case 4 and it is difficult to imagine, if the convulsions had been due to hyponatraemia, that the child could have survived for several days without treatment.

Case 5 is another example of an encephalopathy with trismus, in this case almost certainly due to whooping-cough. Although there is no clear clinical history of pertussis, the blood picture is characteristic, and the histopathological findings are in keeping with the diagnosis of whooping-cough encephalopathy.[21-23] The role of the hypoglycaemia is difficult to assess.

The possibilities of leukaemia, malignant reticulosis or lipoidosis were excluded on histological evidence.

SUMMARY

1. Five cases showing trismus during their fatal illnesses are described.

2. Autopsy findings are available in 4 cases and, in each, lesions were demonstrated microscopically in the mid-pontine tegmentum.

3. The aetiology in these cases was varied, viz:

(*a*) post-vaccinal encephalitis, which is apparently very rare in South Africa, and this case proven pathologically is thought to be the first recorded.

(*b*) polio-encephalitis.

(*c*) purulent meningitis.

(*d*) unknown aetiology, despite careful autopsy.

(*e*) whooping-cough encephalopathy.

4. The pathological findings in 2 of the cases illustrate the differences between 'virus encephalitis' and 'post-infective perivenous demyelinating encephalitis.'

We wish to thank Dr. E. Kahn (Senior Paediatrician), Dr. S. Wayburne (Paediatrician) and the Superintendent of Baragwanath Hospital for permission to publish cases 1-4; Dr. B. Epstein (Paediatrician) and the Superintendent of Edenvale Hospital for permission to publish case 5.

We are indebted to the Director of the South African Institute for Medical Research and Dr. R. Cassell, Dr. J. Higginson and Dr. I. W. Simson, of his staff, for permission to use the pathological material of cases 1, 2 and 4; and Dr. M. M. F. Fitzpatrick for case 5. Mr. M. Ulrich of the Photographic Department was responsible for the photomicrographs.

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FEMALE GENITAL TUBERCULOSIS

A CLINICAL REVIEW OF 62 CASES ENCOUNTERED IN THE GROOTE SCHUUR HOSPITAL DURING THE THREE YEARS 1954-56 *

by

WILLEM H. MULLER, M.B., CH.B., M.O. AND G. (CAPE TOWN) †

Klerksdorp

The problem of female genital tuberculosis has lately received greater recognition, largely owing to the fact that more extensive use is being made of endometrial biopsies. During the past 18 years the finding of unsuspected endometrial tuberculosis in women who are apparently in perfectly good health, and who complain solely of infertility, has become increasingly frequent and should no longer arouse surprise in the investigator. Nevertheless, in South Africa the pre-operative diagnosis of genital tuberculosis is still relatively rarely made. The most probable reason for this is that it is not suspected frequently enough, particularly in cases presenting with infertility or menstrual anomalies or resistant chronic pelvic infection. An attempt will be made in this review to compare the clinical aspects of the present series with well known and authoritative series published from various centres in the world, e.g. Scotland, England, Scandinavia, the United States of America, Israel and Chile, in order to support the contention that genital tuberculosis should be suspected more frequently in this country.

* Based on an address delivered to the Cape Western Subgroup of the South African Society of Obstretricians and Gynaecologists at Cape Town on 10 December 1956.

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Material: In 1954 Moore[1] reviewed 26 cases of genito-peritoneal tuberculosis admitted to the gynaecological wards of Groote Schuur Hospital during the 5-year period 1949-53. The present report is based on 62 cases of genital tuberculosis seen in the same hospital during the 3-year period 1954-56. The number of beds was increased in 1954.

INCIDENCE

The incidence of all types of genital tuberculosis admitted to the gynaecological wards is shown in Table I and is compared with those from other centres. From this table it is apparent that the percentage incidence of cases seen during the past 3 years is double that seen during the preceding 5 years, and is the same as that obtaining in Glasgow in a recent 20-year period.

Racial Incidence. There were 49 Coloured patients, 10 Bantu patients and 4 White patients in the present series. The racial incidence is set out in Table II, from which it is apparent that during the period 1949-56 genital tuberculosis was 7 times more frequent in the non-White patients than in the White patients. The greatest incidence was in the Bantu (8½ times as frequent as in the White patients). As genital tuberculosis is accepted as a secondary infection

TABLE I. INCIDENCE OF GENITAL TUBERCULOSIS IN GYNAECOLOGICAL ADMISSIONS

Author	*Hospital*	*Period*	*Total Gynaecological Admissions*	*No. of Cases Genital Tuberculosis*	*Incidence %*
Sutherland (1951) ..	Royal Samaritan, Glasgow ..	20 years	65,943	369	0·56
Schaeffer (1956)	New York Lying-in	23 years	32,823	44	0·13
Moore (1954)	Groote Schuur	5 years (1949-53)	9,239	26	0·28
Muller (1956)	Groote Schuur	3 years (1954-56)	10,963	62	0·57
Groote Schuur (1949-56)	Groote Schuur	8 years (1949-56)	20,202	88	0·44

TABLE II. RACIAL INCIDENCE OF GENITAL TUBERCULOSIS (GROOTE SCHUUR HOSPITAL) 1949-56

Author	*White*			*Coloured*			*Bantu*			*Total non-White*		
	Gynaec. Admissions	*No. of Genital Tuberc.*	*Incidence %*	*Gynaec. Admissions*	*No. of Genital Tuberc.*	*Incidence %*	*Gynaec. Admissions*	*No. of Genital Tuberc.*	*Incidence %*	*Gynaec. Admissions*	*No. of Genital Tuberc.*	*Incidence %*
Moore 1949-53	4,501	4	0·1	4,112	18	0·43	626	4	0·64	4,738	22	0·5
Muller 1954-56	4,015	4	0·1	5,941	48	0·8	1,007	10	0·99	6,948	58	0·83
Total 1949-56 ..	8,516	8	0·1	10,053	66	0·65	1,633	14	0·85	11,686	80	0·68

from a previous pulmonary infection in the vast majority of cases, it will be revealing to record the pulmonary tuberculosis notification rate per 1,000 population in Cape Town

TABLE III. PULMONARY TUBERCULOSIS NOTIFICATION RATE (PER 1,000 POPULATION)

Centre	*Year*	*White*	*Non-White*	*Total*
Cape Town ..	1949-1955	0·94	5·2	3·1
Durban	1954	—	—	3·38
Johannesburg ..	1954	—	—	1·82
Pretoria	1955	0·17	1·94	1·1
Glasgow	1954	—	—	2·03
Scotland	1954	—	—	1·38
England	1954	—	—	0·84
Stockholm	1954	—	—	0·18

and to compare this with similar rates from other centres in the world (Table III). An approximate estimation may thus be obtained of the incidence of genital tuberculosis that may be expected in these different centres. From this

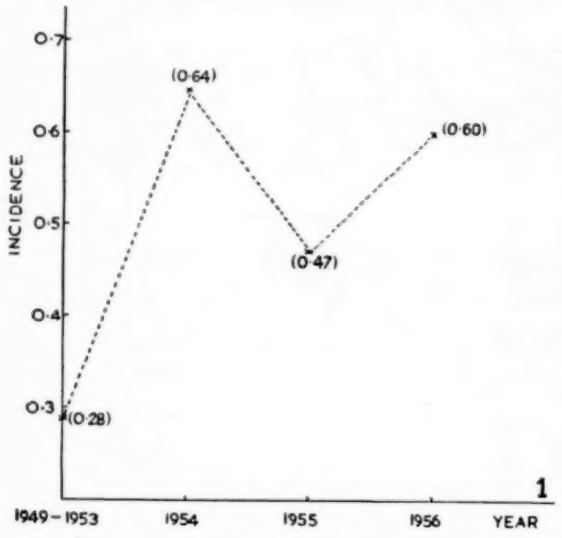

Fig. 1. Incidence of genital tuberculosis as percentage of total gynaecological cases admitted.

table it becomes apparent that pulmonary tuberculosis is 5½ times more common in non-White patients in Cape Town—the same argument holds with regard to the relative incidence of genital tuberculosis, as shown in Table II. Furthermore, it is apparent that the incidence of pulmonary tuberculosis is higher in Cape Town and Durban than in any of the other centres mentioned. As such, it could be expected that the incidence of genital tuberculosis should be correspondingly higher likewise.

TABLE IV. INCIDENCE OF GENITAL TUBERCULOSIS PER YEAR IN GROOTE SCHUUR HOSPITAL (1949-53)

Year	*No. of Gynaec. Admissions*	*No. of Genital Tuberc.*	*Incidence %*
1949-53	9,239	26	0·28
1954	2,492	16	0·64
1955	4,032	19	0·47
1956	4,439	27	0·60
Total, 1949-56	20,202	88	0·44

Incidence of Genital Tuberculosis per year. Fig. 1 and Table IV show the incidence of genital tuberculosis per year in the total gynaecological admissions to Groote Schuur Hospital during the years 1949-56.

Frequency of Site Affected. In the present series the sites involved are shown in Table V. This compares with reported

TABLE V. FREQUENCY OF SITE AFFECTED

Site	*No. of Cases*	*%*
Endometrium	43	69
Tubes	14	22
Cervix	6	9
Genito-peritoneal	5	8
Vulva	1	1·6
Vagina + Urethra	1	1·6
Myometrium	1	1·6

series where the site of the lesion, as found at operation, is as shown in Table VI. The tubes are involved in 90 to 100% of all cases of genital tuberculosis and careful examination will reveal the disease to be bilateral in most cases. Schaeffer[8] states that the endometrium is involved in at least 50% of cases, the ovaries in about 25%, cervix 3%, vulva and vagina 1%. In the present series tuberculous endometritis was found on 43 occasions, 40 cases being diagnosed by curettage or biopsy of the endometrium and 3 cases from hysterectomy specimens. The incidence of tuberculous endometritis found on curettage is 0·36% of gynaecological admissions. This compares with Sutherland's[9] incidence of 0·27% in 200 cases reported. In Sutherland's series (over a 20 year period) the incidence of endometrial tuberculosis was more than 3 times as common in the second 10-year period as in the first—the probable reason being the more extensive use of endometrial study in various conditions, particularly infertility. The discovery of tuberculous endometritis on curettage means for practical purposes that the tubes are involved as well, whether or not they are palpably enlarged (Stallworthy[10], Haines,[11] Novak[12]). Tubal involvement may, however, be present in the absence of endometrial involvement, as the endometrium is said to be affected in about 50% of cases of tuberculous salpingitis. Tuberculous salpingitis is responsible for 5-10% of all forms of salpingitis. The incidence of genital tuberculosis in surgically removed adnexa is shown in Table VII.

TABLE VI. SITE OF LESION AS FOUND AT OPERATION

Author	*No. of Cases*	*Tubes %*	*Uterus %*	*Ovaries %*	*Cervix %*	*Vagina %*	*Peritoneum %*
Greenberg (1921) ..	200	100	45	29	3·5	0·5	63
Wetterdal (1924) ..	56	100	66·6	55	—	—	45
Norris (1928)	33	90	50	40	2	2·0	—
Jedberg (1950) ..	186	100	24·7	18·8	—	—	32·8
Total	475	99	40	28·8	3·2	0·7	48

TABLE VII. INCIDENCE OF GENITAL TUBERCULOSIS IN SURGICALLY REMOVED ADNEXA

Author	*No. of Cases of Genital Tuberculosis*	*Total No. of Pathological Specimens*	*% Genital Tuberculosis*
Greenberg (1921) ..	200	2,959	6·76
Solomons (1923) ..	53	390	14·0
Wetterdal (1924) ..	42	290*	14·5
Norris (1928)	—	—	7·3
Williams (1948) ..	7	91	7·7
Vieria (1949)	33	225†	14·7
Novak (1952)	—	—	5·0
Total	335	3,955	8·5

* Cases of Salpingitis.
† Patients with Pelvic Inflammatory Disease.

AETIOLOGY

Family History of Tuberculosis. About 20% of patients with genital tuberculosis give a history of tuberculosis in their immediate family.[16] As a rule they were exposed to the infection in their childhood. Barnes[17] has shown that there seems to be a special vulnerability of the female genital tract to invasion by the tubercle bacillus when the primary infection (in the lung in the great majority of cases) coincides with the period of adolescence or early maturity, i.e. approximately at the age of the menarche.

Previous Health. Barnes found amongst 107 patients that 73% showed evidence of other forms of tuberculosis but in only 13% was the extragenital lesion still in an active state. The vast majority (80%) had evidence of a previous pulmonary lesion. The incidence of extragenital tuberculous manifestations has also been reported in patients suffering from genital tuberculosis by Clayton[18] (36%), Jedberg[7] (56%), Schaeffer[3] (47%) and Sutherland[9] (56%). It is therefore obviously of importance to note in the history such manifestations as pleurisy, peritonitis, erythema nodosum,

TABLE VIII. RADIOLOGICAL PULMONARY INVESTIGATION 41 CASES

Lesion	*No. of Cases*	*%*
Active Tuberculosis	4	10
Previous Tuberculosis	12	29
Total Tuberculosis	16	39
Lungs Clear	25	61

glandular, renal or osseous tuberculosis, or pulmonary disease. The records of the present series allow the estimation of the incidence of extragenital tuberculosis in 48 patients. In 41 patients radiological investigation of the lungs was performed, the results being recorded in Table VIII. Thus in 39% of cases radiological evidence of pulmonary tuberculosis existed. The incidence of pulmonary lesions in patients with genital tuberculosis recorded in other parts of the world is shown in Table IX. In the present series, 6 cases also had evidence of tuberculous peritonitis at laparotomy and 1 case had an old tuberculous spinal lesion.

TABLE IX. INCIDENCE OF PULMONARY LESIONS IN PATIENTS WITH GENITAL TUBERCULOSIS

Author	*No. of Cases*	*% Pulmonary Tuberculosis*
Jedberg (1950)	186	24
Liljedahl (1950)	148	49
Barnes (1955)	57	80
Schaeffer (1956)	44	29
Total	435	40
Muller (1956)	41	39
Total	476	40

TABLE X. AGE INCIDENCE IN GENITAL TUBERCULOSIS (62 CASES)

Age	*No. of Cases*
19	3
20–30	36
31–40	13
41–50	4
51–60	3
61–70	2
71	1
20–40	49 (79%)

Thus 23 patients out of 48 had evidence of extragenital tuberculosis, an incidence of 48%.

Age Incidence. The age incidence in the present series is shown in Table X. The average age was 25·7 years. This prevalence of the disease during the child bearing years is borne out by figures presented by a number of authors.

TABLE XI. AGE INCIDENCE IN GENITAL TUBERCULOSIS

Author	*No. of Cases*	*Age*	*%*
Greenberg (1921) ..	200	20–40	74
Berry (1940)	50	20–40	90
Vieira (1949)	33	20–40	93
Jedberg (1950)	186	20–40	80
Total	469	20–40	79·4
Groote Schuur Hospital (1949–56)	88	20–40	80
Total	557	20–40	79·4

Marital Status. In the present series 7 patients were single (average age 22 years) and 55 were married, of whom 42 complained of infertility (76%). Primary infertility was the main symptom in 34 patients and secondary infertility in 8.

Previous Pregnancies. Only 17 cases had previously been pregnant, i.e. 30% of the married women. Of these 5 were post-menopausal and 12 were still in the child-bearing age. The most recent pregnancy in the patients in the child-bearing age was 6 months before the diagnosis of genital tuberculosis. This occurred in 2 patients, the pregnancies having terminated in a ruptured tubal ectopic in one patient and an abortion in the other. Data concerning previous pregnancies are recorded in Table XII.

TABLE XII. PREVIOUS PREGNANCIES—17 CASES

Pregnancy	*No. of Cases*
1 Abortion	2
2 Abortions	1
1 Ectopic	1
1 Full Term	3
2 Full Term + 1 Abortion ..	1
2 Full Term	2
3 Full Term	3
4 Full Term	1
5 Full Term	1
10 Full Term	1
13 Full Term	1
Total	17

Sutherland[9] states that only 23 out of a total of 176 patients with genital tuberculosis had had previous pregnancies. In 4·5% of cases in his series, the tuberculous lesion had probably developed during pregnancy or in the postpartum period. In not one of our patients was this found. Sutherland[20] reviewed the literature of postpartum genital tuberculosis. Recent reports of such cases are those of Donaldson,[21] Morrison and Ealand,[22] and McInroy and Craig.[23] Schaeffer[16] states that 28 cases of postpartum and post-abortal genital tuberculosis have been reported in the literature since 1940; 12 were associated with abortion, 9 with premature deliveries and 7 with full-term pregnancies.

SYMPTOMATOLOGY

Menstrual History. It is usually stated that amenorrhoea is a rare symptom of genital tuberculosis.[3, 7, 9, 18, 24] Having been associated with the gynaecological endocrine service at Groote Schuur Hospital for the past 3 years, where amenorrhoea, oligomenorrhoea and hypomenorrhoea figure prominently in the symptomatology of cases seen, frequently in association with genital tuberculosis, I find the statement of the rarity of amenorrhoea surprising. The menstrual histories given by our patients are recorded in Table XIII. Of the 19 cases with amenorrhoea, 3 were of the primary type and 16 of the secondary type. The significant feature is the fact that in no fewer than 70% of cases there were either amenorrhoea, oligomenorrhoea or hypomenorrhoea. The probable significance of this discrepancy between the present series and recorded incidences elsewhere will be discussed under symptomatology.

TABLE XIII. MENSTRUAL HISTORY IN GENITAL TUBERCULOSIS 62 CASES

Menses	*No. of Cases*	%
Oligomenorrhoea and/or Hypomenorrhoea	25	40
Amenorrhoea	19	30
Menorrhagia; Metrorrhagia	10	16
Regular	11	18
Dysmenorrhoea	19	30
Post-Menopausal Bleeding	3	5
Menopausal	4	7

Table XIV records the symptomatology of large series of cases recorded previously and this may be compared with the symptomatology in the present series. The marked discrepancy in the amenorrhoea, oligomenorrhoea and hypomenorrhoea incidences is obvious.

Infertility was the commonest symptom in the present series (76%). The association between endometrial tuberculosis and infertility is now well established—reviews of the literature have been published by Sutherland,[9, 25] Sharman,[26] Schaeffer,[8] and Studdiford.[27] The incidence of genital tuberculosis amongst patients attending infertility clinics averages 5% (Table XV)—demonstrated by routine curettage or endometrial biopsy. However, since tuberculous endometritis is present in only about 50% of cases of tuberculous salpingitis, one may assume that many infertile patients have tuberculous salpingitis but present a negative endometrial biopsy.[16] It would appear that 10% is a more accurate figure for the incidence of genital tuberculosis in infertility patients. Since the establishment of an infertility clinic at Groote Schuur Hospital in 1955, 355 patients have attended. Tuberculous endometritis was discovered in 6 cases, an incidence of 1·6%. This, however, is not the true incidence since curettage or biopsy was performed in 90 cases only, and the incidence of tuberculous endometritis in these 90 cases is 6·6%—probably much nearer the true incidence. It is of fundamental importance to realize that often the *only* complaint in women with genital tuberculosis is infertility. This was the case in 50% of Stallworthy's series[24] and 47% in Liljedahl's report.[18] In the present review only 5% of cases complained solely of infertility. The reason for this small incidence is probably the relatively small number of cases complaining of infertility who were subjected to curettage or biopsy. The necessity for *routine* study of the endometrium in all cases complaining of infertility is amply borne out by the figures. Furthermore, it is contended that a considerably higher incidence than 1·6% will be obtained in the infertility clinic at Groote Schuur Hospital should this procedure be adopted in future.

TABLE XV. INCIDENCE OF GENITAL TUBERCULOSIS IN INFERTILITY PATIENTS

Author	*No. of Patients*	% *Genital Tuberculosis*
Jedberg (1950)	1,168	2·1
Liljedahl (1950)	499	10·0
Rabau (1950)	2,000	3·5
Haines (1952)	200	4·0
Sharman (1952)	2,985	5·2
Halbrecht (1953)	2,000	5·0
Total	8,852	4·7
Groot Schuur Hospital (1955-56)	355	1·6
Groote Schuur Hospital (1955-56)	90 (D & C Biopsy)	6·6

TABLE XIV. SYMPTOMATOLOGY IN GENITAL TUBERCULOSIS (PER CENT)

Author	*No. of Cases*	*Infertility* %	*Abdominal Pain* %	*Dysmenorrhoea* %	*Menorrhagia* %	*Amenorrhoea* %	*Oligomenorrhoea and/or Hypomenorrhoea* %
Greenberg (1921)	200	—	80	62	41	6	24
Jedberg (1950)	186	35	51	2	21	1	—
Liljedahl (1950)	148	47	20	20	9	3	12
Stallworthy (1952)	78	57	50	21	—	—	10
Sutherland (1956)	200	39	26	—	17	11	—
Schaeffer (1956)	44	43	50	5	36	7	—
Total	856	42	46	26	24	10	17
Muller (1956)	62	76	38	30	16	30	40

Abdominal Pain. Chronic hypogastric pain is a common symptom in genital tuberculosis. Usually the pain is not severe and is of long duration. Episodes of acute pain have generally been attributed to secondary infection by pyogenic organisms.[16] In the present review abdominal pain was present in 38% of cases (24 patients). In 4 patients the onset was acute—in 2 due to ruptured ectopic pregnancy in tuberculous tube; in 1 a tubal abortion was found and in 1 the symptomatology and findings suggested an acute abdominal catastrophe; however, a generalised genito-peritoneal tuberculosis was found.

Menorrhagia and Metrorrhagia. These symptoms were present in 16% of cases in the present series. Abnormal uterine bleeding has been reported in 10-40% of patients with genital tuberculosis.[16] Stallworthy[24] regards this as the commonest menstrual anomaly.

Amenorrhoea, Oligomenorrhoea, Hypomenorrhoea. The period of amenorrhoea varied from 3½ months to 12 years, the average being 2½ years. Oligomenorrhoea was defined as infrequent menses occurring at intervals of not longer than 3 months. These symptoms were present in no fewer than 70% of our patients which, as noted above (Table XV), is at complete variance with the reported incidence in world literature. The probable reason for this discrepancy is that genital tuberculosis was suspected (particularly in the latter part of the period under review) in all cases presenting with these symptoms; had the same suspicion obtained in cases presenting with infertility or excessive menstrual bleeding or in the resistant chronic pelvic infection cases, the relative incidence of cases complaining of amenorrhoea, oligomenorrhoea or hypomenorrhoea and diagnosed as genital tuberculosis might have been less, since the others would therefore have been greater. Nevertheless, the symptoms of amenorrhoea, oligomenorrhoea and hypomenorrhoea should always evoke the strongest suspicion of genital tuberculosis, especially when they are associated with a clinical picture which otherwise resembles either 'chronic pyogenic or gonococcal pelvic infection' or even 'acute pelvic infection' or 'ovarian' or 'tubo-ovarian cysts'.

Dysmenorrhoea, usually of the congestive type, was present in 30% of cases in this series. The reported incidence varies from 2 to 62%, and Schaeffer[16] believes that it is not more common in patients suffering from tuberculosis than in the non-tuberculous person.

Miscellaneous Symptoms. In the present series the following miscellaneous presenting symptoms were noted: vaginal discharge (3 cases), post-menopausal bleeding (3 cases), lump in the abdomen (2 cases), post-coital bleeding (1 case), dyspareunia (1 case) and genital prolapse (1 case). A history of poor general condition over a period of months or years associated with weight loss, undue fatigue, low-grade persistent fever and vague lower abdominal pain is frequently elicited in these patients.[16] In the present series complaints of general disability were noticeably infrequent.

PHYSICAL SIGNS

The physical findings in cases of internal genital tuberculosis may show considerable variation, from apparent normality on pelvic palpation to signs suggestive of chronic adnexal disease with thickened adnexa, unilateral or bilateral tubal or tubo-ovarian masses of restricted mobility, or others suggestive of a mobile 'ovarian' cyst or even an 'acute abdomen'. The finding of what is thought to be an inflammatory adnexal mass (especially if it is unilateral) in a patient with a suggestive history should always arouse the suspicion of genital tuberculosis. Similarly, elicitation of adnexal inflammatory masses in a virgin makes the exclusion of tuberculous pelvic disease mandatory. The physical signs in this series are tabulated in Table XVI. In Sutherland's series of 200 cases[9] no fewer than 36% had no palpable abnormality in the pelvis. As a rule the adnexal masses are less tender in patients with tuberculous disease than in those with pyogenic infections. It is of significance that in 19 out of 27 cases the adnexal mass was unilateral. This does not signify that the opposite adnexa are not involved as, microscopically, tubal involvement is bilateral in 100% of cases.[10]

TABLE XVI. PHYSICAL FINDINGS IN GENITAL TUBERCULOSIS, 62 CASES

Finding	*No. of Cases*	
Adnexal Masses	27	
	Unilateral	19
	Bilateral	8
R/V Uterus Only	11	
Fibroids	4	
Ulceration of Cervix	4	
Acute Abdomen	4	
Miscellaneous	6	
Pelvis N.A.D.	6	

DIAGNOSIS

The pre-operative diagnosis of genital tuberculosis should rest largely on suspicion, with confirmation forthcoming from the use of the biopsy curette, full curettage or bacteriological investigations. Hysterosalpingography may be used as an ancillary method in diagnosis. In the present report the clinical diagnoses on admission are listed in Table XVII.

TABLE XVII. CLINICAL DIAGNOSIS ON ADMISSION, 62 CASES

Clinical Diagnosis	*No. of Cases*	*Tuberculosis suspected*	*Tuberculosis should have been suspected*
Amenorrhoea, Oligomenorrhoea; Hypomenorrhoea—for investigation	22	16	6
Infertility for Investigation	11	4	7
? Cancer for Investigation	7	0	2
Ovarian Tumour	6	0	4
Fibroid	5	0	2
Ectopic Pregnancy	4	0	0
Chronic Pelvic Infection	2	0	1
Miscellaneous	5	0	1
Total	62	20	23

Thus, while genital tuberculosis was suspected in only 32% of cases in this review, it should have been suspected in at least 69%. The cases where it should have been suspected were mostly those where the significance of the association of the symptoms of amenorrhoea, oligomenorrhoea or hypomenorrhoea with the clinical diagnosis of 'ovarian cysts' or chronic pelvic infection or infertility or fibroids went unheeded. Under these circumstances it is of fundamental importance to exclude tuberculosis. The association of endometrial tuberculosis with the symptoms of infrequent or scanty menses, which we have seen so frequently of late,

has naturally aroused the suspicion in any case whose menstrual pattern is of that type. At the gynaecological endocrine clinic for practical purposes, we have, in the presence of these symptoms, been accustomed to exclude the following conditions: (1) Genital tuberculosis, (2) ovarian hyperthecosis, (3) obesity and its variants, (4) psychogenic and occupational causes.

In the present series the diagnosis was established by curettage in 37 cases and by endometrial biopsy in 3 cases. The indications for endometrial study in these cases are set out in Table XVIII. The steady increase in the number of cases diagnosed by endometrial study is amply borne out in this table. This was mostly due to the fact that cases

TABLE XVIII. INDICATIONS FOR ENDOMETRIAL STUDY, 40 CASES

Indication	1954	1955	1956	*Total*
Amenorrhoea, Oligomenorrhoea, Hypomenorrhoea	5	4	13	22
Infertility	0	4	7	11
Menorrhagia, Metrorrhagia ..	1	1	2	4
Chronic Pelvic Infection ..	0	1	0	1
? Cancer	0	2	0	2
Total	6	12	22	40

seen at the infertility clinic and endocrine clinic were investigated by curettage. There has, generally speaking, also been an *increased awareness* recently of the necessity of more often suspecting genital tuberculosis in gynaecological cases seen at Groote Schuur Hospital. In 1957 it is intended to establish a pelvic infection clinic and it is hoped that thereby even fewer cases of genital tuberculosis will remain undiagnosed. This clinic will, incidentally, also serve as a follow-up clinic for cases of tuberculous pelvic disease. Table XIX records the method of diagnosis in the whole series.

TABLE XIX. METHOD OF DIAGNOSIS IN GENITAL TUBERCULOSIS 62 CASES

Method	*No. of Cases*
Curettage or Endometrial Biopsy	40
Macroscopic + Biopsy at Laparotomy	6
Hysterectomy Specimen	5
Tubal Specimen	5
Vaginal, Vulval Biopsy	2
Miscellaneous	4

As a further aid to facilitate diagnosis in future, the records were scrutinized to obtain an idea what previous treatment or investigations these patients had received. Five cases had inadequate records; 24 cases were investigated immediately or soon after first attending and this led to the diagnosis; 6 cases were treated in the Medical Department for headaches (3 cases) and miscellaneous ailments, e.g. diabetes, myxoedema (3 cases). The remaining 27 cases were treated in the Gynaecological Department and on analysis presented the following information: 11 cases had one or more courses of short-wave diathermy to the pelvis; 6 cases were investigated for infertility without a curettage being done; 5 cases received various gynaecological treatments, e.g. ventrisuspension, dilatation of the cervix, pessary for retroversion, cervical cautery; 4 cases received empirical oestrogen therapy for the symptom of amenorrhoea; 1 case was observed for the symptom of secondary amenorrhoea. This analysis once more bears out the need for, (1) investigation of the resistant case of chronic pelvic infection, (2) routine endometrial study in infertility cases, and (3) routine endometrial study in cases presenting with menstrual anomalies (especially if with infrequent or scanty menses).

Bacteriological Diagnosis. The final diagnosis of tuberculosis rests upon the demonstration of the tubercle bacillus cultured on suitable media or obtained from guinea-pig inoculation or, more rarely, seen in direct smears from discharges. The use of bacteriological methods not only results in the discovery of unsuspected cases of genital tuberculosis, but may also confirm the diagnosis which could not be made with certainty from histological examination alone. Repeated examinations may be necessary and one negative bacteriological or histological result certainly does *not* rule out the possibility of a tuberculous lesion. In this series material from 17 cases was sent for simultaneous histological and bacteriological examinations (16 endometrial curettings and one vaginal biopsy). The results in these 17 cases are recorded in Table XX and compared with series reported elsewhere. In the present series guinea-pig inoculations were made on 12 occasions, 7 being positive and 5 negative. Kirschner culture was done on 13 occasions, 6 being positive and 7 negative. In only one case was the bacteriological result positive whilst the histological result was negative.

Radiological Investigation of the Genital Tract. In the present series hysterosalpingography has only been an ancillary aid in diagnosis. Magnusson[30] states that tuberculous pelvic disease presents distinctive features on hysterosalpingography. Whether these features should be accepted as diagnostic in the absence of confirmatory histological or bacteriological evidence remains undecided. It is in the patient where, in spite of repeated negative histological and bacteriological investigations, the clinical suspicion of genital tuberculosis still obtains, that it would greatly facilitate the diagnosis of the condition if the diagnostic value of hysterosalpingography were accepted. A number of such patients are being followed up in Groote Schuur Hospital.

TABLE XX. COMPARISON OF HISTOLOGICAL AND BACTERIOLOGICAL INVESTIGATION

Author	*No. of Cases*	*No. of Cases Positive Histology*	%	*No. of Cases Positive Bacteriology*	%
Liljedahl (1950)	105	103	98	84	80
Halbrecht (1953)	46	23	50	46	100
Sutherland (1956)	185	185	100	141	76
Total	336	311	92	271	80
Muller (1956)	17	16	94	8	47

CONCLUSIONS

It is contended that this clinical review allows the following conclusions to be reached:

1. Genital tuberculosis should be suspected much more frequently.
2. It should be suspected especially in patients complaining of infertility or menstrual anomalies or resistant chronic pelvic infection, and more particularly so if there is a suggestive history.
3. In taking the history of patients with gynaecological complaints, the possibility of previous manifestations of tuberculosis should always be remembered.
4. Routine curettage or endometrial biopsy is mandatory in cases presenting with infertility or menstrual disturbances (especially amenorrhoea, oligomenorrhoea and hypomenorrhoea) or resistant chronic pelvic infection.
5. In Cape Town and Durban especially, the incidence of pulmonary tuberculosis is very high and consequently it could be expected that the incidence of genital tuberculosis should be correspondingly high.
6. An increased awareness of the possibility of genital tuberculosis in cases seen in Groote Schuur Hospital has resulted in the diagnosis being made more frequently.

SUMMARY

1. A clinical review of 62 cases of genital tuberculosis seen in Groote Schuur Hospital during the 3-year period 1954-56 is presented and is compared with series from different centres in the world.

2. It is contended that genital tuberculosis should be suspected much more frequently in South Africa, especially in Cape Town and Durban.

I gratefully acknowledge the interest shown by Professor James T. Louw and all my colleagues who referred cases for investigation. I should like to thank the statistical departments of the Cape Town City Corporation and the Groote Schuur Hospital for the data supplied and Dr. N. H. G. Cloete, Medical Superintendent, Groote Schuur Hospital, for permission to publish this review. The histological and bacteriological work was done by members of the Department of Pathology of the University of Cape Town, to whom I am most grateful.

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CHOLELITHIASIS AND FLUORINE

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New York

In the present state of our knowledge biliary calculi are believed to be composed of cholesterin, calcium bilirubin or biliverdin, and traces of calcium carbonate. Their formation is attributed to stagnation and inspissation of bile in a biliary system in which a catarrhal congestion had set in as a result of extension of bacterial invasion from the bowel. Thus a mild cholecystitis develops, leading to a breakdown of cells in the mucous membrane. Both the cholesterin and the calcium, which were first believed to be due to normal secretion from the blood, are now looked upon as the product of this cell disintegration. It is assumed that the calcium combines with bilirubin to form a precipitate acting as a nucleus on which cholesterin is deposited.

An analogy suggested itself in the mechanism of formation of biliary calculi, on the one hand, and of urinary-tract calculi, on the other. Since in the development of urinary calculi fluorine, by virtue of its forming a substantial constituent in their chemical composition, has been found in many cases to play a vital role (Spira, 1956), it became a matter of great interest to investigate whether similar conditions might also apply in the formation of biliary calculi.

MATERIAL

Biliary calculi removed by operation from 10 patients taken at random were submitted to chemical analysis for fluorine. The following is a summary of reports on findings obtained by Dr. H. Amphlett Williams, Ph.D. (Lond.), A.C.G.F.C., F.R.I.C., Public Analyst, London, England:

CONCENTRATION OF FLUORINE IN BILIARY CALCULI EXPRESSED IN PARTS PER MILLION (P.P.M.)

	No.	Marking	Age	Sex	Fluorine
1	.. ..	BH 287078	56	m	9
2	.. ..	SR	42	m	3
3	.. ..	DE 1	46	f	7
4	.. ..	DE 2	56	m	10
5	.. ..	ESN	86	f	32*
6	.. ..	SAB 1	not known		1
7	.. ..	SAB 2	not known		5
8	.. ..	EA	54	f	<2
9	.. ..	PS	53	m	3
10	.. ..	MV	46	f	4

* Repeat analysis carried out by Analytical Chemists, Cincinnati, Ohio, revealed a concentration of 40 p.p.m. of fluorine.

DISCUSSION

The first studies of the action of fluorine on experimental animals were carried out as late as the end of the nineteenth century. Schulz 1889, found hyperaemia in the liver and other internal organs in several of his animals. All the fluorine administered perorally to a dog by Brandl and Tappeiner (1891) was, in the first stages of the experiment, retained in the body. As accumulation increased, it was excreted principally in the urine and to a much lesser extent in the faeces, the quantity excreted with the faeces being estimated at not more than between 1/10th and 1/5th of the quantity excreted with the urine. A part of the fluorine daily administered continued to be deposited, most likely as calcium fluoride, mainly in the skeleton and the teeth, in quantities of 5·19% and 1% respectively, as compared with the amounts ingested and excreted. In the liver, the third organ in the order of fluorine retention, 0·59% was deposited. The quantity retained in other organs was not determined.

More recently, the effect of ingested fluorine on the liver has been examined at autopsy by a few investigators. Schwyzer (1903), quoted by DeEds (1933), reported congestion and hydropic and fatty degeneration of the liver in acute fluorine poisoning and fatty degeneration alone in chronic. Chaneles (1929) found congestion of the liver, Sharkey and Simpson (1933) advanced cloudy swelling, and Rabinowitch (1945) extensive and severe parenchymatous changes accompanied by deposits of readily recognizable crystals of calcium fluoride in the liver, kidneys, and other organs in a case of acute fluorine poisoning. Phillips and Lamb (1934), however, recorded less constant changes in the liver than in the kidneys of their experimental rats, and Kick (1930), quoted by Phillips and Lamb, observed no effect of fluorine on the liver. Biester, Greenwood and Nelson (1936) reported that in dogs receiving fluorine in doses comparable with those encountered in some water supplies over a period of 7-12 months histological changes were found in the small intestine but none in the stomach and gall-bladder; in the liver, no inflammatory or infiltrative changes were detected, though varying degrees of cloudy swelling and hydrops were observed, which were not always correlated with the fluorine level administered. Roholm (1937) found nothing definitely abnormal on microscopic examination of the livers of experimental pigs. The livers of calves and dogs showed no macroscopic or important microscopic structural changes, but the cell protoplasm exhibited signs of degeneration. At the post-mortem examination of one cryolite worker, the liver showed normal shape and size, but there was considerable congestion and the surface was slightly irregular. Microscopically, pronounced stasis of the organ was found, and the liver cells were greatly vacuolated (fatty degeneration?). In another cryolite worker, the liver was slightly diminished in size and its surface was somewhat wrinkled and thickened in places, but there was no change in its consistency. In the gall-bladder system nothing abnormal was detected. It will be remembered that cryolite, a fluorine mineral of the composition $Na_3\ AlF_6$ (or 3NaF. AlF_3), is an indispensable raw material employed in the manufacture of aluminium.

In normal cattle, which were not experimentally exposed to fluorine, Gautier and Clausmann (1913) found that fluorine was present in all organs and tissues, though in variable amounts. In the bile of cattle there was 0·13 mg. of fluorine per 100 g. of the fresh, and 1·03 mg. per 100 g. of the dry substance. In a normal man, who was 57 years old at the time of a fatal accident, 0·64 mg. of fluorine per 100 g. was found in the fresh liver, and 2·13 mg. per 100 g. in the dried organ. Chang, Phillips, Hart and Bohstedt (1934) reported traces of fluorine in all the normal tissues of dairy cattle studied. As compared to bone, the liver was relatively free of fluorine, containing less than 1 mg. per 100 g. of the dried normal tissue.

The daily ingestion of drinking water with a fluorine concentration as low as 1 p.p.m., or higher, during the period of calcification of the permanent teeth, that is to say, during the first 8 years of life, produces mottling of the teeth as the first external visible sign of chronic fluorine poisoning (fluorosis). As the daily ingestion of fluorine in quantities of 1 mg. or more continues, further signs and symptoms develop which, in the last analysis, are brought about by the halogen depriving the body of calcium, material indispensable for sustaining the vitality of most of the organic functions of the body (Starkenstein, 1923).

All the patients here recorded were living in areas whose drinking water was ascertained to contain only insignificant traces of fluorine. It is therefore obvious that the halogen found in their gall-stones was derived from sources other than drinking water, namely, from articles of food ingested in normal every-day diet, practically all of which are known to be contaminated by variably large amounts of the poison contained in preservatives, sprays, insecticides, fungicides, rodenticides, chemical fertilizers, and so forth, widely employed in the food industry. In the process of preparing food in aluminium cooking utensils, acids and alkalis contained in, or added to, the food corrode the metal so as to liberate fluorine and other impurities present in it and to contaminate the food. According to McClure (1939), there is a greater retention of fluorine experimentally administered to rats in food than when equal quantities were ingested in drinking water.

Case 5 in the series here recorded seems to be of special interest, since the fluorine content of 32 p.p.m. and 40 p.p.m. respectively detected in fragments of the gall-stone analysed was considerably higher than that found in any of the other cases. When the patient died in 1919, a lump which for years had been palpated at the side of her abdomen was by her last wish examined and found to contain a number of gall-stones. For sentimental reasons they were preserved by three generations, and fragments were now submitted to specific analysis for fluorine. The analytical chemist reported that they consisted chiefly of cholesterol.

The study of the action of fluorine and the knowledge of the symptomatology of chronic fluorine poisoning are of but recent origin. The pathological changes brought about by the protracted ingestion of fluorine are not yet sufficiently well established, and the number of reports at hand is as yet too small to serve as a basis on which final conclusions can be drawn. To complicate matters, the few reports in existence do not tally, probably because for some as yet unknown reason fluorine does not act consistently in every case, but concentrates its attack sometimes on one organ, and sometimes on another. This may well explain why, even in animal experiments, the biliary system, with which we are here primarily concerned, was not uniformly found to be affected.

In chronic fluorine poisoning, the mucous membranes appear to be frequently involved. Damage to the submucosa of the urinary bladder, manifested by occasional small foci of eosinophiles and mild vacuolization, has been observed by Biester, Greenwood and Nelson (1936), and Spira (1928, 1944, 1946), reported that conjunctivitis, stomatitis, gingivitis, pharyngitis, and catarrh of the upper air passages are often encountered in the disease picture of fluorosis.

It is submitted that, being a protoplasmatic poison, it is primarily the fluorine rather than the microorganisms derived from an infected bowel that damages the mucous membrane of the gall-bladder, the bacterial invasion being of a secondary nature. The detritus of disintegrated cells of the mucous membrane will thus serve to form the future nucleus around which, owing to its affinity to and its precipitating action on calcium, that part of the fluorine ingested which finds its way into the gall-bladder combines with the normal calcium content of the tissues to produce calcium fluoride as one of the components of the calculi. This development is probably facilitated by the anatomical position of the gall-bladder which, together with the damaged and congested mucous membrane of the biliary system, causes stagnation of bile and continued precipitation of calcium salts and cholesterin.

Whilst up to this point there appears to be a remarkable similarity in the mechanism of the formation of biliary calculi and of urinary calculi respectively, there is a wide discrepancy between their fluorine contents, which requires elucidation. Against the average fluorine concentration of 449 p.p.m. (or 562 p.p.m.) found in 10 urinary calculi (Spira, 1956) the average fluorine concentration in 10 biliary calculi here recorded amounted to not more than 7·6 p.p.m. In other words, 59 (or 74) times as much fluorine was on the average found in urinary as in biliary calculi taken at random.

Further investigation will be required to find the reason for this incongruity. For the present, the fact must be kept in mind that, whereas with urinary calculi the mineral constituents are derived from a fluid medium, with biliary calculi they are absorbed by the viscous bile to combine with cholesterin. It is a well-known fact that, with the present imperfect method of analysis, the determination of the fluorine content in an organic medium is fraught with great difficulty.

Whatever the explanation, the fact that fluorine ingested with normal every-day food reaches the gall-bladder and becomes part, however slight, of biliary calculi may throw some additional light on the problem of cholelithiasis.

SUMMARY

Biliary calculi removed from 10 patients living in areas whose drinking water was fluorine-free were found to contain fluorine in concentrations ranging between 1 p.p.m. and 32 (or 40) p.p.m. It was concluded that the fluorine was derived from articles of normal everyday food, practically all of which are known to be contaminated by variably large amounts of the poison contained in preservatives, sprays, insecticides, fungicides, rodenticides, chemical fertilizers, and so forth, widely employed in the food industry, as well as from food prepared in aluminium cooking utensils, in the manufacture of which the fluorine mineral cryolite is an indispensable raw material.

The role played by fluorine in the formation of biliary calculi is discussed.

ZUSAMMENFASSUNG

In Gallensteinen von 10 Patienten, deren Trinkwasser fluorfrei war, wurde Fluor in Konzentrationen nachgewiesen, die zwischen 1 Teil Halogen auf 1,000,000 Teile Konkrement und 32 (bzw. 40) Teilen Halogen auf 1,000,000 Teile Konkrement schwankten. Fluor ist in Nahrungsmitteln des täglichen Lebens als Verunreinigung enthalten und gelangt in den Körper auf dem Wege über Konservierungsmittel, in der Gartenpflege gebrauchte insektenvertilgende und pilzzerstörende Substanzen und Obstbaumspritzmittel, sowie über chemische Düngmittel, die in der modernen Landwirtschaft vielfach benützt werden.

In Aluminiumkochgeschirr zubereitete Speisen nehmen im Kochen das Fluor vom Metall auf, das sowohl von Säuren als auch von Alkalien leicht angegriffen wird. In der Herstellung des Metalls wird das fluorhaltige Kryolit als unumgänglich notwendiges Rohmaterial verwendet.

Die Rolle, die das Fluor in der Bildung von Gallensteinen spielt, wird besprochen.

RÉSUMÉ

Dans les calculs biliaires de 10 malades, qui buvaient de l'eau démunie de fluor, on a pu déceler le fluor en quantités de 1 part sur 1 million parts de calculs à 32 (ou 40) parts sur 1 million parts de calculs. Le fluor se trouve dans les produits alimentaires comme impureté et passe dans l'organisme par les produits de conservation, des insécticides, des engrais chimiques, des produits de conservation pour les arbres, qui sont utilisés largement dans l'agriculture. Les pots d'aluminium peuvent être attaqués par les acides, ainsi que par les alcalis, et les aliments cuits dans ces pots absorbent le fluor qui est contenu dans l'aluminium préparé à base de cryolithe.

Le rôle du fluor dans la formation des calculs biliaires est discuté.

OPSOMMING

Dit is gevind dat galstene wat verwyder is van 10 pasiënte van wie die drinkwater vry van fluor was, van 1 tot 32 (of 40) dele fluor per miljoen dele bevat. Die gevolgtrekking is gemaak dat die fluor in die galstene van voedsel afkomstig is, waarvan byna alles met hierdie stof besmet is, omdat fluor-bevattende bederfwerende, insekdodende, swamdodende, en knaagdierdodende stowwe, chemiese bemestingstowwe, ens. op 'n groot skaal in die voedselindustrie

gebruik word. Die fluor is waarskynlik ook gedeeltelik afkomstig van die aluminiumgereedskap wat in die bereiding van voedsel gebruik word.

Die rol wat fluor in die ontstaan en opbou van galstene speel, word bespreek.

I wish to express my gratitude to all those who kindly supplied me with gall-stones from various sources.

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RESOLUTIONS AND STATEMENT BY THE COUNCIL FOR THE PHARMACEUTICAL TRADE AND INDUSTRY

The following resolution was passed at a meeting of the Council for the Pharmaceutical Trade and Industry at Cape Town on 6 April 1957:

This Council

(*a*) recognizing that it is in the interest of the public and of the pharmaceutical profession to conform to the principle stated by the South African Medical Council that doctors should not place themselves in economic competition with chemists and druggists, accepts as a policy that the supply of all pharmaceutical and ethical products to doctors, dentists and veterinarians shall be at a nominal handling discount only off the consumer prices except in cases where there is no chemist and druggist in business within a radius of 5 miles.

(*b*) resolves that a reasonable nominal handling discount to doctors, dentists and veterinarians is 10% off the established consumer price. If there is no established consumer price, the price to the doctor, dentist and veterinarian shall be trade price plus 35%. In areas where there is no chemist and druggist in business within a radius of 5 miles, the discount shall be 25% or the price shall be trade price plus 10%.

(*c*) Agrees that the policy adopted in this resolution be implemented as from 1 July 1957.

(*d*) Agrees that the Council may, in its discretion, at the request of any person or firm, exempt from any pharmaceutical and/or veterinary preparation from the operation of this resolution.

STATEMENT

The pharmaceutical distributive system in South Africa comprises:

(i) Importers, Manufacturers and/or Primary Distributors.
(ii) Wholesale Distributors, and
(iii) Retail Distributors.

Selling price structures for both the distributive sections (wholesale and retail) as well as consumer groups, are set against basic prices determined by the manufacturers, importers and/or primary distributors. Assessment of the place accorded to each distributive section and the ultimate consumer price allows each distributive section what has been traditionally accepted over many years as an economic margin to cover the particular services rendered by each section in the distributive system.

It is in the public and national interest, and necessary for the effective functioning of the distributive trade, that these established margins and price structures are maintained so as to ensure full facilities at all times for distribution with equal facility to all sections as well as to the public. This has developed into a system which has made pharmaceutical and ethical products available at uniform price levels throughout the country, while still providing the free play of competition so essential from the point of view of public and national interest.

The sale or supply of pharmaceutical and ethical products to the public by doctors, dentists and veterinarians in the course of, or associated with, dispensing practices has come within the purview of the South African Medical and Dental Council which, in January 1956, confirmed that it was undesirable for doctors to place themselves in economic competition with chemists and druggists. This attitude has been followed up by the pharmaceutical distributive trade independently of any action or activity by other bodies which have dealt with this matter purely on its professional implications, vis-à-vis the respective rights of chemists and druggists and of medical practitioners.

The Council for the Pharmaceutical Trade and Industry was established in June 1955 and includes the recognized organizations of the elements concerned in the production and distribution of pharmaceutical and ethical products. One of the main objects of the Council is to coordinate the activity of these organizations and ensure maintenance of the traditional facilities enjoyed by all the distributive groups.

The Council recently passed a resolution as above, to be implemented from 1 July 1957. It is believed that this resolution will translate into practical terms the attitude of the Medical and Dental Council without prejudice to practitioners who supply medicines, etc., to their patients as a part of medical service without the object of deriving therefrom any economic enhancement of their practice.

It is not believed that the resolution can have the effect of penalizing any member of the public by increasing the legitimate cost of their medicines, nor will it prevent any practitioner from securing such material as may be necessary for special use in his practice at the most competitive price level.

IN MEMORIAM

MARINUS VAN DEN ENDE, M.B., CH.B. (CAPE TOWN), PH.D. (CANTAB), F.R.S.S.AF.

Prof. R. W. James, Vice-chancellor and Acting Principal, University of Cape Town, writes: On 4 June 1957, Marinus van den Ende, since 1946 Professor of Bacteriology in the University of Cape Town, and Dean of the Faculty of Medicine, died after a long illness borne with a courage that was an inspiration to his colleagues. He was a man of great scientific ability. Those of us working in fields quite other than his felt instinctively that here was a man destined to make his mark in the scientific thought of his generation. He was an essentially modest man, even a diffident one when his own work was under discussion; but he was plainly

one of those rare beings, a few of whom one fortunately meets as one goes through life, in whom the real fire burns. His death at the early age of 45 is a loss to medical science not easy to estimate. Others will doubtless speak of this aspect of his life. I am a layman in this field, and I wish to write of another side of his character, and one in which he also showed quite outstanding ability—his powers as a university administrator.

Van den Ende's quick grasp of the essentials of a situation, his moderation, his ability to weigh opposing views, and his fair-mindedness and intellectual honesty, about which there could never be the least doubt, made him an ideal man on those committees in which general university policy is discussed. The late Dr. T. B. Davie was very quick to recognize his quality in this respect, and had the very highest opinion of his powers and his judgment.

When a brilliant young investigator also shows signs of unusual common sense and judgment—and the two things go together not uncommonly, which is after all natural enough—there is a real danger that he will soon be loaded down with committee work, greatly to the detriment of his teaching and research; and it is often very difficult for a man to know where his duty to the University really lies. A man of van den Ende's mould, extremely conscientious, and knowing, as he must have done, for all his innate modesty, something of his powers and the value of his counsel to the University, finds himself being taken more and more from the scientific work that he loves, and which he feels to be the real object of his life, into the difficult, and sometimes troubled, field of university administration and policy.

Van den Ende, I know, felt this at times keenly. He was in charge of a highly successful virus-research unit, and was its guide and inspiration; but he knew how very much more he could have done had he not had the duties imposed on him by the Deanship of the Faculty of Medicine, membership of the University Council, and of the numerous committees and boards which these offices entail. These tasks too he did supremely well, and I believe that he liked doing them, and knew that he was making a real contribution to the well-being of the university he loved. The difficulty of reconciling this division of a man's energies between teaching and research on the one hand, and his wider duties to the university as a whole is only too familiar, and is probably fundamentally insoluble. Van den Ende did more in both fields of his activities than is given to most of us to accomplish in either; but the difficulty became acute when, 4 years ago, he was attacked by a malady that he knew well must prove mortal within a comparatively short span of years. The story of those years is a tale of high courage and devotion to duty, which those who worked with him find it difficult to contemplate without emotion. He decided that he would, to the limit of his powers, carry on with his work and serve the university, and this, quite simply, he did in the time that remained to him.

Towards the end of 1955 it was becoming plain that the duties of the Deanship were more than he could be fairly asked to carry, and it was decided to appoint a new full-time Dean, and that van den Ende should resume his post of Professor of Bacteriology and devote himself so far as possible to his scientific work. While the question of the new Dean was under consideration, and before any suitable appointment could be made, the Principal of the University, Dr. T. B. Davie, died in London. Negotiations between the University and the Province on matters connected with the financing of the Joint Medical Scheme were in a critical state, and many of the rather complicated details were really understood only by Dr. Davie and Professor van den Ende. A newly appointed Dean, possibly from outside the University, would have been in a very difficult position, and van den Ende therefore offered to remain in office as part-time Dean and Professor of Bacteriology, and to give me in my capacity of Acting Principal the benefit of his advice and knowledge in the matters affecting the Medical School, of which I was at the time necessarily still largely ignorant.

I was very worried about this, but I believe now that it was probably the right course; for the feeling that matters were not being correctly handled and that he was not able to use his knowledge of affairs and personalities to keep our negotiations on the admirable and co-operative lines for which they have been notable, would have worried him deeply, and would have been a constant source of trouble and irritation to him.

I find it very hard to express adequately my personal indebtedness and gratitude to van den Ende for his help and counsel during the past 2 years. I hope he knew how much I appreciated it, and how keenly his colleagues in the University and the Hospital valued his devotion to duty and his courage. Van den Ende was greatly loved. Any mention of his name between colleagues was accompanied by that warm tone of affection that one learns to recognize. We felt his innate nobility of mind, and knew that we had been privileged to know a man of quality finer than the ordinary. We mourn a loved friend, but we mourn something more than that, a brave and fine soul.

Prof. G. A. Elliott, of Johannesburg, writes: Professor Marinus van den Ende, Dean of the Faculty of Medicine of the University of Cape Town, Head of the Department of Pathology, Professor of Bacteriology, and a great South African, died after a long illness in Cape Town on 4 June 1957 at the age of 45.

Marinus van den Ende was born in Potgietersrust in 1912, in which town he received his school education, matriculating at the age of 15, then proceeding to the University of Cape Town. After completing a year of house appointments at the new Somerset Hospital, Cape Town, he accepted a post in his University's Department of Pathology under Professor B. J. Ryrie. At the time he was uncertain whether he would ultimately follow a career in the clinical field or in the field of pathology. Under the tutelage and encouragement of Professor Ryrie, he became intensely interested in the morbid anatomical aspects of pathology, and his work during the 3 years that he held his appointment was recognized as being of a sufficiently high standard for him to be offered a research fellowship to proceed to Cambridge University in 1937 to work in Pathology under Professor A. N. Drury. His work at Cambridge was his introduction to serious full-time research. For the work which he carried out there on anaphylaxis, he was later awarded the degree of Doctor of Philosophy of that University. Whilst at Cambridge, the high quality of his work attracted the attention of Sir Henry Dale, then Director of the Institute for Medical Research, Hampstead, London, where he was given an appointment at the conclusion of his research fellowship. With his customary modesty, he felt that he was achieving little, but he was nevertheless learning the research method the hard way under the strict discipline for which the Institute is famous. He was working at the time on 'Reverse Anaphylaxis'. At the Institute he was posted to work under the late Sir Patrick Laidlaw and Dr. W. H. Andrews on the study of viruses. He studied their nature and was also concerned with the production of influenza virus vaccine which was being prepared against a threatening pandemic of influenza.

Soon after the outbreak of World War II, he led a research team studying the control of the spread by air of infection in hospitals, showing that the bacterial content of air was greatly reduced if fusel oil was applied to floors and to dust-trapping fabrics such as blankets and sheets. When typhus became a major problem in the campaign in North Africa, he was flown there as a member of a research team to study that disease, and followed the fighting forces to Naples, where typhus was rife. His work with the team later appeared in a booklet published by the Medical Research Council of Great Britain. Thereafter he returned to London to continue his studies in Virology.

In 1946 he accepted the post of Professor of Bacteriology at the University of Cape Town. On the retirement of Professor B. J. Ryrie in 1948 he became Head of the Department of Pathology. He rapidly build up in that department a virus research team, and his personal ability in this field received official recognition when the Council for Scientific and Industrial Research established under his directorship their Virus Research Unit. He continued his studies on the nature of viruses until he was compelled by his failing health to abandon his scientific work only a few weeks before his death. He contributed to the study of the poliomyelitis virus, and discovered the cause of lumpy skin disease. By now he was a world authority on viruses.

In 1953, he was awarded a Sims Research Fellowship, and proceeded to Australia to work in the laboratory of Sir MacFarlane Burnett. His period of research under Burnett was cut short by the onset of the illness which eventually caused his death, and he had to return to South Africa.

In 1954, he was appointed Dean of the Faculty of Medicine of the University of Cape Town. His interest and ability in the administrative field became as intense and effective as it was in the field of his scientific studies, and he was responsible for developing a very close cooperation between the Cape Provincial

Administration authorities administering the teaching hospital at Groote Schuur, and the University. He was also instrumental in introducing many advances in the medical educational sphere.

As Dean of the Faculty, he represented the University of Cape Town on the South African Medical and Dental Council and became the very active chairman of the Council's Medical and Dental Education Committee. His last administrative meeting 3 weeks before his death took place at his home, where he was confined to bed very ill, the object of the meeting being to collate the numerous opinions that had been received by the South African Medical and Dental Council from South Africa and the rest of the world on standards of medical education.

He took an active part in the administrative affairs of the Council for Scientific and Industrial Research, and at the time of his death was chairman of the Medical and Dental Advisory Committee of that body.

He was a member of the technical advisory committee of the Poliomyelitis Foundation, and of various other Committees and Commissions concerned with health services for the community.

In 1955 he was invited to a conference sponsored by the Ciba Foundation in London, and delivered a paper on his virus researches.

As a teacher, he inspired his students with a sense of seeking after the truth. Owing to failing health, he had to give up most of his teaching in the last 2 years of his life.

No one had a greater sense of duty and a greater sense of seeking after truth. He had an outstanding capacity to study a problem, whether scientific or administrative, grasp it in its totality and in its perspective in relation to other problems, and then to expound on the problem with brilliant incisiveness and clarity so that all who heard were in no doubt of his meaning and in no doubt of the implications of the problem. Sensitive inwardly to criticism to an extreme degree, he nevertheless spoke his views and acted according to his sound judgment, fearless of what criticism he might receive. His integrity and sense of justice, right and duty, were the guiding lights of his short life.

From the time that the diagnosis of lymphadenoma was made in 1953, the mental anguish which he suffered was hidden from all but those who knew him intimately. The communion of silence between himself and his intimate friends was his solace. His greatest support through these difficult years was the wonderful understanding of his wife. He faced the world during these last years with aggressive courage, and far from relaxing from the arduous duties of his multiple tasks, he made greater efforts than ever to complete in the few remaining years allotted to him the work which he had set himself to do in a normal lifetime.

In 1939 he married Joan Herold Barry, daughter of Mr. Richard John Barry, then Master of the Cape Supreme Court. She and their 4 children, Jan, Pieter, Joan Ida and Marina, survive him. To them we extend our heartfelt condolences.

Of no one could it be more aptly said: 'Let us not grieve because he is dead, but rather let us rejoice because he lived.'

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent Fund during January, February and March 1957, are gratefully acknowledged.

Votive Cards in Memory of:

Dr. J. D. Allen *by* Dr. A. J. Orenstein, Mr. and Mrs. D. S. Haddon, Dr. H. Kaye, Medical Graduates Association, Johannesburg, Mr. J. A. Chambers, Mr. and Mrs. D. J. Laing and Family, Mr. Dalling, Inspector of Mines, 14 of his colleagues at Germiston, Mrs. Alma Campbell, Miss Gwen Edwards, Mrs. J. B. Bullen, Dr. and Mrs. C. G. Booker, Director and Staff, S.A.I.M.R., Medical Officer of Health and Staff of City Health Department, Miss Margot Macfarlane, Dr. W. G. McDavid, Dr. and Mrs. McCartney.

Dr. M. Orford *by* Dr. L. I. Braun and Dr. C. F. Krige.

Dr. J. W. Rabkin *by* Dr. F. Krone, Dr. A. W. Sichel, Dr. J. S. du Toit and Dr. P. W. J. Keet.

Mrs. Dr. A. Berg *by* Dr. F. Krone and Dr. A. W. Spratt.

Mr. R. O. Goolden *by* Dr. F. O. Fehrsen.

Mr. T. J. Mostert *by* Dr. G. M. Malan.

Dr. J. I. Lipschitz *by* Dr. F. Krone.

Total amount received from Votive Cards, £49 16s. 6d.

Services Rendered to:

Ashley Furman *by* Dr. S. N. Javett.

Dr. D. H. Vollet *by* Drs. H. B. Savage, H. Grant-White and E. G. Wilbraham.

Mrs. Dr. Macleod *by* Dr. N. A. Rossiter and Dr. Lockwood Mundy.

Mr. Franks *by* Dr. Pat Lynch.

Mrs. A. Enslin *by* Dr. Maurice Weinbren.

Dr. P. G. Large *by* Dr. B. W. Franklin Bishop.

Mrs. Irene Fairbairn *by* Dr. D. Standing.

Mrs. Vandeyer *by* Dr. Naiker.

Mrs. M. Bailey *by* Dr. T. D. G. Fairbairn.

Mrs. B. Kavalsky *by* Drs. Finlayson and Clegg.

Total Amount received from Services Rendered, £34 17s. 0d.

Donations:

	£	s.	d.		£	s.	d.		£	s.	d.		£	s.	d.
Members of Cape Western Branch Collection Box ..	4	18	6	Dr. H. O. Hofmeyer	0	5	6	Dr. J. A. V. van Zyl	0	10	6	Dr. M. H. Campbell	0	10	6
				Dr. R. D. Osler ..	1	0	0	Dr. R. W. Nash ..	0	10	6	Dr. N. Campbell ..	0	10	6
				Dr. H. F. Snyman	0	15	0	Dr. N. B. H. Veldsman	0	10	6	Dr. C. A. V. Ovendale	0	10	6
Members of Cape Western Branch Collection Box ..	3	1	1	Dr. F. K. te Water-Naude	1	1	0	Dr. F. A. van Heerden	0	10	6	Dr. J. D. Napier ..	0	10	6
				Dr. D. J. Cilliers ..	1	1	0					Dr. J. R. Reznek ..	0	10	6
Prof. E. C. Crichton	1	0	0	Dr. E. Krausey ..	0	10	6	Dr. C. C. Brown ..	0	10	6	Dr. J. Walker ..	0	10	6
Dr. R. Lipschitz ..	0	10	6	Dr. J. C. Schoeman	0	10	6	Dr. L. Brown ..	0	10	6	Dr. M. E. Schiele ..	0	10	6
Dr. I. Waynik ..	1	1	0	Dr. M. J. Cohen ..	1	1	0	Dr. H. A. Edwards	0	10	6	Dr. B. Birch ..	0	10	6
Dr. S. A. Lange ..	0	10	6	Dr. G. P. J. van Niekerk	1	0	0	Dr. J. K. McKechnie	0	10	6	Dr. E. L. Galgut ..	0	10	6
Dr. G. C. Linder ..	0	10	6					Dr. D. L. Ranking..	0	10	6	Dr. A. S. Nethercott	0	10	6
Mr. R. L. Forsyth..	1	1	0	Dr. W. C. J. Cooper	1	0	0	Dr. E. H. Boodrie ..	0	10	6	Dr. M. Minde ..	0	10	6
Dr. T. I. Hardy ..	2	2	0	Dr. H. F. Schiller ..	0	10	6	Dr. D. G. Cowie ..	0	10	6	Dr. J. H. Symington	0	10	6
Dr. J. Morris ..	1	1	0	Dr. B. Epstein ..	1	1	0	Dr. R. J. Marot ..	0	10	6	Dr. C. Gordon ..	0	10	6
Dr. R. W. H. Welsh	0	10	6	Dr. L. Impey ..	0	10	0	Dr. E. Cassim ..	0	10	6	Dr. J. Smithers ..	0	10	6
Dr. H. E. Bernstein	0	10	6	Dr. E. C. Greenfield	0	5	6	Dr. W. R. Phillips ..	0	10	6	Dr. T. A. Seekings	0	10	6
Dr. M. G. Wolff ..	1	1	0	Dr. W. I. Robertson	0	10	0	Dr. B. A. Armitage	0	10	6	Dr. J. R. Grieve ..	0	10	6
Dr. R. Glasser ..	0	16	6	Dr. G. M. Malan ..	1	1	0	Dr. D. S. Palmer ..	0	10	6	Dr. S. J. Adendorff	0	10	6
Dr. J. W. Harris ..	1	0	0	Dr. Brian M. Porter	1	1	0	Dr. R. G. de Kock	0	10	6	Dr. J. D. M. Barton	0	10	6
Dr. A. J. Ballantine	0	9	6	Dr. F. R. Luke ..	0	10	6	Dr. M. Ginsberg ..	0	10	6	Dr. A. I. Pitcher ..	0	10	6
Dr. M. Myers ..	1	1	0	Dr. A. Asherson ..	0	10	6	Dr. D. H. Biggs ..	0	10	6	Dr. T. H. Crough ..	0	10	6
Dr. J. P. de Villiers	0	10	0	Dr. G. C. Cruywagen	0	5	6	Dr. J. O. Harle ..	0	10	6	Dr. J. P. Beazley ..	0	10	6

	£	s.	d.
Dr. J. Joseph ..	0	10	6
Dr. R. B. Peckham	0	10	6
Dr. S. H. Daneel ..	0	10	6
Dr. L. J. van Wyk	0	10	6
Dr. J. C. Brown ..	0	10	6
Dr. H. A. Kalley ..	0	10	6
Dr. E. C. Morley ..	0	10	6
Dr. J. F. Taylor ..	0	10	6
Dr. F. J. Bennett ..	0	10	6
Dr. K. M. Williams	0	10	6
Dr. S. S. Hoffman	0	10	6
Dr. A. E. Pinneger	0	10	6
Dr. A. R. Bain ..	0	10	6
Dr. N. M. Thompson	0	10	6
Dr. E. Frankenfeld	0	10	6
Dr. C. Akerman ..	0	10	6
Dr. M. T. S. Conradie	0	10	6
Dr. H. H. Stormanns	0	10	6
Dr. N. A. A. van Buuren	0	10	6
Dr. C. C. Akerman	0	10	6
Dr. R. Krikler ..	0	10	6
Dr. B. Krikler ..	0	10	6
Dr. F. S. Drewe ..	1	0	0
Dr. R. P. Stafford ..	1	1	0
Dr. S. C. O'Molony	1	1	0
Dr. A. T. F. Maske	1	1	0
Dr. R. Resnekov ..	1	1	0
Dr. I. Goldberg ..	1	1	0
Dr. J. S. Smit ..	0	5	0
Dr. L. Tomory ..	1	1	0
Dr. L. Sive ..	0	10	6
Dr. E. C. A. Fristedt	1	1	0
Dr. H. M. Fyvie ..	1	1	0
Dr. A. G. Sweetapple	0	10	6
Dr. E. Tucker ..	0	10	0
Dr. F. G. Shearing	1	1	0
Dr. F. Krone ..	1	1	0
Dr. W. J. Perkins ..	0	10	6
Dr. K. P. Haslop ..	1	1	0
Dr. J. van Schalkwyk	0	10	6
Dr. C. D. Scott ..	0	10	6
Dr. P. D. G. Quirke	0	7	6
Dr. B. J. Brewitt ..	0	10	6
Dr. R. L. Baikie ..	0	10	6
Dr. J. F. Rivers-Moore	0	10	6
Dr. L. R. Tibbit ..	0	10	6
Dr. W. Fabian ..	0	10	6
Dr. A. Nel	0	10	6
Dr. R. R. MacKenzie	0	10	6
Dr. R. T. Vaughan	0	10	6
Dr. H. Lowenstein	0	10	6
Dr. L. Fernley ..	0	10	6
Dr. M. A. Lloyd ..	0	10	6
Dr. D. S. McCall ..	0	10	6
Dr. P. S. Bell ..	0	10	6
Dr. J. de V. Meiring	0	10	6
Dr. J. Kleinman ..	0	10	6
Dr. G. W. Moggridge	0	10	6
Dr. N. C. Hopkins	0	10	6
Dr. D. F. Symonds	0	10	6
Dr. C. C. Haupt ..	0	10	6
Dr. P. N. Smith ..	0	10	6
Dr. F. E. Bamford	0	10	6
Dr. T. H. Whitsitt	0	10	6
Dr. L. M. Cohen ..	0	10	6
Dr. H. Levin ..	0	10	6
Dr. W. Blignaut ..	0	10	6
Dr. J. R. Clacey ..	0	10	6
Dr. J. S. Barnes ..	0	10	6
Dr. J. L. Parker ..	0	10	6
Dr. N. K. Pein ..	0	10	6
Dr. E. van Wyngaard	0	10	6
Dr. C. G. Williams	0	10	6
Dr. H. Simon ..	0	10	6
Dr. N. M. L. Lund	0	10	6
Dr. V. A. van der Hoven	0	10	6
Dr. H. P. Militz ..	0	10	6
Dr. C. L. L. Murray	0	10	0
Dr. P. Hack ..	0	10	0
Dr. S. Stein ..	0	10	6
Dr. J. D. Woods ..	0	10	6
Dr. P. V. MacGarry	0	10	6
Dr. A. L. Wilson ..	0	10	6
Dr. L. G. MacKenzie	0	10	6
Dr. S. H. Cohen ..	0	10	6
Dr. W. E. McLelland	0	10	6
Dr. S. Kahn ..	0	10	6
Dr. A. D. Harfman	0	10	6
Dr. L. H. Botha ..	0	10	6
Dr. C. R. Hallot ..	0	10	6
Dr. H. S. Edwards	0	10	6
Mr. J. A. Currie ..	1	1	0
Dr. J. A. Gill ..	0	10	6
Dr. S. H. Kaplan ..	1	0	0
Dr. W. F. Leith ..	1	1	0
Dr. R. Cowley ..	0	10	6
Dr. D. R. van Rooyen	0	10	6
Dr. M. Maister ..	0	10	
Dr. P. T. Hauff ..	0	10	
Dr. D. A. C. Kelly	0	10	6
Dr. I. E. Meyer ..	0	10	6
Dr. H. I. Maister ..	2	2	0
Dr. C. A. Marais ..	1	1	0
Dr. N. Z. Sacks ..	0	10	6
Dr. J. G. Richter ..	0	10	6
Dr. R. A. Findlay	0	10	6
Total	£207	9	7

BOOKS RECEIVED RECENTLY IN THE WITWATERSRAND MEDICAL LIBRARY*

Anderson, W. A. D. Synopsis of pathology. 4 ed. London. Kimpton. 1957.

Association for Research in Nervous and Mental Disease. Neurologic and psychiatric aspects of the disorders of aging. Baltimore. Williams & Wilkins. 1956.

Blount, J. P. Fractures in children. Baltimore. Williams & Wilkins. 1954.

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Ciba Foundation. Colloquia on endocrinology. Volume 10. London. Churchill. 1957.

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Jonas, G. Handbook on horticultural therapy. Hastings, Mich. Ptd. by Hastings Banner. 1955.

Jones, F. W. Trends of life. London. Arnold. 1953.

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Lipman, B. S. Clinical unipolar electrocardiography. Chicago. Yearbook publishers. 3rd ed. 1956.

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Medical Research Council. The hazards to man of nuclear and allied radiations. London. H.M.S.O. 1956.

Moore, F. D. Metabolic response to surgery. Springfield. Thomas. 1952.

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Newcastle Regional Hospital Board. Use of colour in hospitals. Newcastle. 1955.

Pfeiffer, J. Human brain. London. Gollancz. 1955.

Pumphrey, R. J. Origin of language. Liverpool U.P. 1951.

Radin, P. World of primitive man. New York. Schumann. 1953.

Riley, C. M. Living with a child with familial dysautonomia. New York. Dysautonomia Association. 1956.

Rogers, C. R. Psychotherapy and personality change. Chicago. University of Chicago press. 1954.

Simpson, G. G. Meaning of evolution. London. Oxford UP.. 1950.

Skinner, B. F. Science and human behaviour. New York. Macmillan. 1953.

Smith, H. W. Principles of renal physiology. New York. Oxford U.P. 1956.

Smout, C. F. V. Gynaecological and obstetrical anatomy and functional histology. 3 ed. London. Arnold. 1953.

Waart, A. de Het levenswerk van Willem Einthoven. Haarlem. Nederlandsch Tijdschrift voor Geneeskunde. 1957.

Whitla, W. Dictionary of medical treatment. 9th ed. London. Baillière.

* Members of the South African Medical Association who use the facilities of the Witwatersrand Medical Library are invited to send in details of books which they would like the Library to acquire.

THE MEDICAL ASSOCIATION OF SOUTH AFRICA

BALANCE SHEET 31ST DECEMBER, 1956

1955 £		£ s. d.	£ s. d.
	Accumulated Funds		
32,409	Balance, 31st December, 1955 ..	33,724 0 6	
1,315 (*Surplus*)	Less; Excess of Expenditure over Income for the Year	2,937 15 5	
33,724		30,786 5 1	
—	Add: Transfer of National Health Services Emergency Fund ..	257 2 6	
			31,043 7 7
	Funds—Capital Accounts		
257	National Health Services Emergency Fund		—
252	*Dr. H. A. Moffat Memorial Fund* ..		287 2 2
243	Balance, 31st December, 1955 ..	252 4 0	
9	Contributions Received during the year	34 18 2	
	Liabilities		
5,507	Sundry Creditors		10,117 0 7
£39,740			£41,447 10 4

1955 £		£ s. d.	£ s. d.
	Fixed Assets		
7,279	Landed Property—'Byrness', Newlands, Avenue, Newlands, Cape—At Cost		7,278 18 9
3,290	Office Furniture, Fixtures and Machines—Head Office and Johannesburg agency		3,928 0 0
	Net Book Value		
3,195	1st January, 1953	3,195 0 0	
1,132	Purchased since that date (At Cost) less Sales (At Book Value)	2,205 13 2	
4,327		5,400 13 2	
1,037	Depreciation since 1st January, 1953	1,472 13 2	
	Investments		
15,635	*Sundry Investments—At Cost* ..		18,905 0 0
4,335	Quoted Union Government Stock (Market Value, 31st December, 1956. £4,145—1955. £4,155)...	4,335 0 0	
9,550	Unquoted Shares	12,820 0 0	
1,750	First Mortgage Bond	1,750 0 0	
	Current Assets		
8,108	Sundry Debtors, less Provision for Doubtful Debts (£600—1955. £600)		7,274 13 8
4,919	Cash on Savings Account, Bank Current Account and on Hand ..		3,773 15 9
	Funds		
257	National Health Services Emergency fund Cash at Bank		—
252	Dr. H. A. Moffat Memorial Fund Cash at Bank		287 2 2
£39,740			£41,447 10 4

INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDED 31ST DECEMBER, 1956

1955 £		£ s. d.	£ s. d.
20,427	*Printing of Medical Journal* ..		21,216 11 3
937	*Printing of Laboratory and Clinical Medicine*		1,086 16 5
23,831	*Administration and Publication Expenses*		25,584 4 3
18,427	Salaries, Pension Fund, Unemployment Insurance and Pension ..	18,853 2 0	
1,241	Sundry Expenses	1,721 7 11	
1,060	Rent	1,524 15 0	
1,107	Postages and Telegrams	1,074 17 6	
798	Printing, Stationery and Office Requisites	765 19 4	
336	Wrappers	703 0 0	
365	Depreciation of Office Furniture, Fixtures and Machines	436 1 6	
297	Telephones	305 1 0	
200	Audit Fees	200 0 0	
4,682	*General Expenses*		4,453 2 9
	Travelling Expenses	4,058 18 3	
2,716	Delegates 3,014 1 9		
617	Staff 1,044 16 6		
450	Overseas Trip—Secretary .. —		
238	Entertainment Allowances.. ..	275 0 0	
17	Expenses—'History of Medicine in South Africa'	65 10 6	
109	Medals	53 14 0	
531	Booklets and Questionnaires ..	—	
4	Bad Debts	—	
2,218	*Expenses—Public Relations Office Johannesburg*		1,389 11 9
500	*Grants to Universities for Library Services*		800 0 0
—	*Special Grant to Southern Transvaal Branch*		500 0 0
250	*Increase in Provision for Doubtful Debts*..		—
£52,845			£55,030 6 5

1955 £		£ s. d.	£ s. d.
34,938	*Income from Medical Journal* ..		33,427 19 8
33,199	Advertising, less Commission ..	31,630 6 0	
1,654	Non-Members' Subscriptions ..	1,652 5 4	
85	Miscellaneous	145 8 4	
838	*Income from Laboratory and Clinical Medicine*		656 16 8
570	Subscriptions	520 2 1	
265	Advertising, less Commission ..	107 5 10	
3	Miscellaneous	29 8 9	
10,954	*Members' Subscriptions*		10,975 12 1
3,349	*Agency Income*..		2,481 16 7
4,081	*General Income*		4,550 6 0
3,067	Insurance Commission	3,377 7 4	
613	Interest on Investments	666 17 6	
281	Miscellaneous	353 8 8	
120	Rent, less Expenses 'Byrness' ..	152 12 6	
1,315 (*surplus*)	*Excess of Expenditure over Income transferred to Accumulated Funds* ..		2,937 15 5
£52,845			£55,030 6 5

THE MEDICAL ASSOCIATION OF SOUTH AFRICA

BENEVOLENT FUND

BALANCE SHEET 31ST DECEMBER, 1956

1955 £		£ s. d.	£ s. d.
	Accumulated Funds		
41,984	Balance, 31st December, 1956 ..	43,712 15 4	
£41,984		£43,712 15 4	

1955 £		£ s. d.	£ s. d.
	Assets		
	Investments at Cost		
7,067	*Union Government Stocks (Quoted)* (Market Value 31st December, 1956—£6,609)		7,067 10 0
(6,609)	£2,500 3½% 1962/65 ..	2,450 0 0	
	£1,500 3½% 1952/57 ..	1,492 10 0	
	£1,125 3% 1957/64 ..	1,125 0 0	
	£1,000 3% 1959/69 ..	1,000 0 0	
	£1,000 3% 1960/70 ..	1,000 0 0	
28,550	*Shares in Building Societies (Unquoted)* .. .. Saambou (Permanente) Bouvereniging—4,626 Fully Paid-up Indefinite Shares of £1 each		4,626 0 0
3,500	*Secured Loan* .. .. .. Medical House (Proprietary) Limited—First Mortgage on Medical House, Wale Street, Cape Town		29,239 0 0
			40,932 10 0
435	*Sundry Debtors*		948 18 0
431	Interest Accrued	948 18 0	
4	Medical Association of South Africa	—	
2,432	*Cash on Savings Bank Account and at Bank*		1,831 7 4
£41,984			£43,712 15 4

INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDED 31ST DECEMBER, 1956

1955 £		£ s. d.	£ s. d.
2,437	*Benevolent Payments*	3,119 16 0	
23	*Stationery and General Expenses* ..	3 1 8	
£2,460		£3,122 17 8	

1955 £		£ s. d.	£ s. d.
1,836	*Interest on Investments*		2,148 13 9
624	*Appropriation from Capital for Additional Benevolence*		974 3 11
£2,460			£3,122 17 8

ACCUMULATED FUNDS

1955 £		£ s. d.	£ s. d.
624	*Appropriation to Income and Expenditure Account for Additional Benevolence*	974 3 11	
41,984	*Balance, 31st December, 1956* ..	43,712 15 4	
£42,608		£44,686 19 3	

1955 £		£ s. d.	£ s. d.
40,664	*Balance, 31st December, 1955* ..		41,984 5 9
1,944	*Contributions to Capital for the Year ended 31st December, 1956* ..		2,702 13 6
1,128	Donations	2,063 14 0	
573	Services Rendered	365 13 6	
243	Votive Cards	273 6 0	
£42,608			£44,686 19 3

REPORT OF THE AUDITORS TO THE MEMBERS OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA

We have examined the books and accounts and vouchers of the Association and have satisfied ourselves of the existence of the securities. We have obtained all the information and explanations which, to the best of our knowledge and belief, were necessary for the purpose of our audit. In our opinion, proper books of account have been kept by the Association, so far as appears from our examination of those books.

The above Balance Sheet and attached Income and Expenditure Account are in agreement with the books of account. In our opinion, and to the best of our information and according to the explanations given to us, the said Accounts give the information required by the Companies Act 1926, as amended, in the manner so required and the Balance Sheet gives a true and fair view of the state of the Association's affairs as at 31st December, 1956, and the Income and Expenditure Account gives a true and fair view of the deficit for the year ended on that date.

Cape Town
15th March, 1957

Gurney, Notcutt & Fisher
Chartered Accountants (S.A.)
Auditors

BENEVOLENT FUND

We have examined the books and accounts and vouchers of the Benevolent Fund and satisfied ourselves of the existence of the securities. The above Balance Sheet and attached Statements of Income and Expenditure and Accumulated Funds are in agreement with the books of account. In our opinion the Balance Sheet gives a true and fair view of the state of the Fund's affairs as at 31st December, 1956, and the Statements of Income and Expenditure and Accumulated Funds give a true and fair view of the Income and Expenditure of the Fund in respect of the year ended that date.

Cape Town
7th March, 1957

Gurney, Notcutt & Fisher
Chartered Accountants (S.A.)
Auditors.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Maybaker (S.A.) (Pty.) Ltd. announce the introduction of two new medical products and supply the following information:

Sonergan

'Sonergan' is an association of promethazine and butobarbitone, each tablet containing 75 mg. of butobarbitone ('Soneryl') and 15 mg. of promethazine hydrochloride ('Phenergan).

Butobarbitone has been used with success for many years to produce sedation and hypnosis in minor nervous conditions and mild insomnia. Promethazine has been widely used as a potent antihistamine and anti-emetic. Its central sedation effects, characterized by somnolence, relief of mental tension and enhancement of the activity of hypnotics, are being increasingly utilized.

The association of these two effective and clinically established preparations in 'Sonergan', provides adequate and pleasant sedation with a smaller dose of butobarbitone than would otherwise be necessary, and there is in addition relief of anxiety and mental tension.

'Sonergan' is particularly valuable in the pre- and post-operative care of patients because of the considerable anti-emetic properties of promethazine. It is indicated:

(*a*) for general use at bedtime for cases of insomnia (1 or 2 tablets as required);

(*b*) for special use in pre-operative preparation (2 or more tablets).

(*c*) for post-operative sedation (1 or 2 tablets night and morning).

'Sonergan' is presented as sugar-coated tablets in containers of 50 and 500.

Stemetil

'Stemetil' brand 1- [3-(3-chloro-10-phenothiazinyl) propyl]-4-methyl-piperazine dimaleate is a new phenothiazine derivative closely related to 'Largactil'. It resembles 'Largactil' in the range of its pharmacological actions but it shows marked quantitative differences. Thus it is several times more active as an anti-emetic but less active in reducing conditioned and instinctive reflex activity.

Clinical investigations have established that 'Stemetil' is of value in the symptomatic management of:

(*a*) migraine and kindred conditions;
(*b*) Menière's syndrome and other labyrinthine disorders;
(*c*) giddiness of other origins;
(*d*) nausea and vomiting.

In common with other phenothiazine derivatives 'Stemetil' may be useful in the management of psychiatric disorders. This is still being investigated and insufficient evidence is available for definite recommendations to be made.

'Stemetil' is administered orally or in the form of suppositories. It is supplied as 5 mg. tablets in containers of 25 and 250, and as suppositories in boxes of 5 × 25 mg.

PASSING EVENTS : IN DIE VERBYGAAN

South African Society of Obstetricians and Gynaecologists. The annual general meeting of the Cape Town sub-group of this society was held at the Here Sewentien Klub, Cape Town, on 20 May 1957. The following were elected to the committee of the sub-group: Dr. P. Massey, Chairman; Dr. L. J. Abramowitz, Secretary; Dr. J. C. Coetzee and Dr. M. Sandler.

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Radiological Society of South Africa. The office bearers and other members of the Council of the Radiological Society of South Africa for 1957-58-59 are as follows: Chairman Dr. F. McLachlan, Secretary Dr. H. Jackson, Treasurer Dr. C. Komins, Executive Member Dr. M. H. Fainsinger, Members—Prof. J. N. Jacobson, Drs. M. J. Meyer, J. Nel and M. Weinbren, and, *ex officio*, Drs. H. Osler, H. H. Navid and J. H. B. Muller.

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Dr. W. Silber. M.B., Ch.B. (Cape Town), F.A.C.S. (Edin.) has changed his address from National Mutual Buildings, Church Square, Cape Town, to 316 Medical Centre, Heerengracht, Cape Town. The telephones remain unchanged, viz. residence 7-4458, rooms 3-7785; if no reply 7-7727.

Dr. W. Silber, M.B., Ch.B. (Kaapstad), F.A.C.S. (Edin.) het sy adres verander vanaf National Mutual-Gebou, Kerkplein, Kaapstad, na Mediese Sentrum 316, Heerengracht, Kaapstad. Die telefone bly onveranderd, nl. woning 7-4458, spreekkamers 3-7785; indien geen antwoord nie, 7-7727.

Dr. M. Cecil Michelow. The telephone number of the rooms a-1 Admirals Court, Rosebank, of Drs. S. Joel Cohen and A. M. Porter, whose specialist practice in obstetrics and gynaecology Dr. M. Cecil Michelow has joined, is 41-1218, and not as stated in the notice under Passing Events in the *Journal* issue of 8 June, on page 574.

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Dr. Isaac Grayce, M.R.C.P. (Lond.), of Cape Town, returned on 21 June after 3 months' stay in the United States and London during which time he attended various medical centres and medical congresses.

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Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van gedugte epidemiese siektes en poliomiëlitis in die Unie get durende die tydperk 29 Mei-6 Junie 1957.

	Poliomiëlitis				
	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	—	—	—	—	—
Kaapprovinsie ..	4	7	7	—	18
Oranje-Vrystaat ..	—	1	—	—	1
Natal	1	3	—	—	4
Totaal ..	5	11	7	—	23

Pes: Een (1) Naturellegeval builepes in die Vryburg-Afdelingsraadgebied. Laboratorium-bevestiging word afgewag.
Pokkies, Tifuskoors: Geen.

REVIEWS OF BOOKS : BOEKRESENSIES

CARDIOLOGY

Cardiology. 2nd Edition. By William Evans, M.D., D.Sc., F.R.C.P. Pp. ix + 574. 569 Illustrations. £5 4s. 3d. plus 2s. 3d. delivery charge. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1956.

Contents: Preface to Second Edition. Preface to First Edition. 1. History Taking and Physical Examination. 2. Cardiac Enlargement. 3. Heart Sounds and Heart Murmurs. 4. Arrhythmia. 5. Congenital Heart Disease. 6. Pericardial Disease. 7. Myocarditis and some other Myocardial Affections. 8. Endocarditis. 9. Cardio-Aortic Syphilis and Aortic Atherosclerosis. 10. Cardiac Pain. 11. Hypertension and Hypotension. 12. Pulmonary Heart Disease. 13. Heart Failure. 14. The Heart in Endocrine Disorders. 15. Heart Disease and Pregnancy. 16. The Heart in some Miscellaneous Conditions. 17. Other Vascular Diseases and Anomalies. 18. Some Other Aspects of Heart Disease. Index.

In the appraisal of any book the reviewer must consider the circumstances which motivated its writing in the first place. This book was first published in 1948, and in the preface Evans states,

'It is meant to serve the need of the medical student who seeks a more concise treatise of this subject in preparation for a qualifying or higher examination'. The author has accomplished the purpose of his book with a high degree of success.

The second edition of 'Evans' Cardiology' is nearly double the size of its predecessor—this despite every effort to limit discussion of diagnostic procedures which are still under trial, and of therapeutic agents 'which have not yet established an undisputed right to be accepted as routine practice in the treatment of a particular heart affection'.

Like the first, the value of this new edition lies in the directness of his approach and the clarity of his description of physical signs. He resorts extensively to illustrations, which are especially well selected; furthermore the important diagnostic features are clearly numbered or lettered in these illustrations, leaving no doubt in the reader's mind what he intends to demonstrate.

The author is at variance with those who make a routine of using a full complement of chest leads in the electrocardiographic investigation of every patient. He claims that the 4 limb leads I, II, III and IIIR, together with the 3 chest leads CR1, CR4 and CR7, provide adequate electrocardiographic evidence for the evaluation of cardiac diseases. The reviewer does not agree that this particular selection of chest leads is adequate in most instances.

Evans' approach to the diagnosis of congenital anomalies of the heart and great vessels is not in keeping with the views of most modern writers, in that he has adhered to Maude Abbott's classification rather than following a classification based on physiologic and dynamic principles. However, the description of the congenital anomalies is in keeping with the rest of his book—very concise and accurate.

Dr. Evans is not particularly impressed with the value of anticoagulant therapy in acute myocardial infarction, nor is it mentioned in the long-term management of coronary heart diseases. In the management of recurrent pulmonary embolism from phlebothrombosis of the veins of the lower extremities, the author believes that ligation of the large veins in the groin or higher offers the best prospects of success. This is an opinion which will also find opposition in many quarters. In the treatment of acute rheumatic fever, the author considers salicylates to be superior to the steroids.

Apart from its being rather sceptical and somewhat critical regarding certain phases of therapy, this book is highly recommended both for students and clinicians.

T.J.D.

NEUROLOGICAL EXAMINATION

Clinical Examinations in Neurology. By Members of the Sections of Neurology and Section of Physiology, Mayo Clinic and Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota, Rochester, Minnesota. Pp. xviii + 370. 87·50. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: I. The Neurologic History. II. Introduction to Use of Various Forms for Recording History and Results of Clinical Examination. III. General Observations and Order of Procedure. IV. The Cranial Nerves. V. Neuro-ophthalmology. VI. Motor Function—Part I: Central Integration of Motor Function. VII. Motor Function—Part II: Specific Study of Muscle. VIII. Reflexes. IX. The Sensory Examination. X. Mental Function. XI. Language and Motor Speech. XII. Autonomic Function. XIII. Clinical Examinations in Selected Problems of Pain. XIV. Electroencephalography. XV. Electromyography and Electric Stimulation of Peripheral Nerves and Muscle. XVI. Biochemical and Pharmacologic Aids in Neurologic Diagnosis. XVII. Examinations of Cerebrospinal Fluid by Lumbar and Cisternal Puncture. Index.

In a short preface to this book the following words appear: The neurologist practices his specialty against the broad background of general medicine. His contributions to the welfare of the patient stem from his mastery of the clinical neurological examination, and his special knowledge of the anatomy and functions of the nervous system and the diseases which affect it. Mastery of the clinical neurological examination is a necessary acquisition. It can be learned only by doing, but guidance can accelerate the process.' They are quoted because of their obvious importance and because it cannot be stressed too much or too often that neurology should not be the mystery it so often is to students nor should it prove a difficult, and often avoided, territory for the general practitioner. A little basic knowledge of neuroanatomy and neuro-physiology, and the practice of complete neurological examinations in case after case, enabling one to evaluate normal responses and thereby the appreciate variations which are pathological—and the subject becomes a fascinatingly interesting one because it is in essence the application of logic and method to a clinical art.

Up to a point this book serves its purpose in guiding the student to attaining some skill in the technique of neurological examination, but it has its defects. Some of these supposed defects may be only the reflection of a reviewer's bias, such as the excessive use of forms and charts and inventories—perhaps to be expected in a product of the Mayo Clinic. A minor criticism is a comparative lack of correlation with anatomy and physiology, though this is not altogether neglected. On the whole it is a useful book, which can be recommended to the houseman and young practitioner.

S.B.

AUTOBIOGRAPHY

One Doctor in his Time. By Bethel Solomons, M.D., F.R.C.P.I., F.R.C.O.G., M.R.I.A. Pp. 224. 9 Illustrations. 18s. net. London: Christopher Johnson. 1956.

Contents: Early Days. School. Trinity College, Dublin and Medical School. Qualified Doctor. Private Practice. Marriage and Fitzwilliam Square. Master of the Rotunda. The United States. All Work and no Play. President of the Royal College of Physicians of Ireland. Liberal Jewry and Anti-Semitism. Rugby Days. Irish Literary Renaissance. Hunting. Some Professional Problems. My Holiday with Dr. O'Sullivan. Towards the End of the Journey.

Doctor Bethel Solomons has given us a very endearing book in this autobiography, and one turns back the last page filled with deep respect and affection for this famous man, who looks upon life and his fellow men with the modesty and the simplicity of the truly great.

Were one to discuss the book from a purely literary point of view, one would perhaps not be over-enthusiastic, but after some opening moments of irritation, even the most carping critic could not but be disarmed by the personality and transparent honesty of thought of the writer, and be flattered at being taken into his confidence.

For those of us who, like the present reviewer, have had the privilege of knowing Dr. Solomons either as a friend or as a teacher in Dublin's Rotunda Hospital his book is pure delight—a *pot pourri* of reminiscent chuckles and at times nostalgic lumps in the throat. But even the casual reader—whatever his interests—will find more than enough to absorb his attention in this cosy gossip of Dr. Solomons. For this author, unlike the usual run of medical men, has lived a full life of almost unbelievable diversity of interests. As a doctor he is internationally acclaimed. In the field of sport he has captained Dublin University at Rugby and has no less than 10 international caps to his credit. He has always been deeply devoted to the arts and meanders happily on about the many famous people who were his friends, leaving one wishing to hear more and still more.

He is a deeply religious man and a passionate Irish patriot and his devotion to his family gives one a soothing sense of security in these precarious days.

His students revered him as a teacher and were enslaved by his humour and the charm of his person. To-day, wherever medical men foregather who were at the Rotunda during his Mastership, his name in one way or another never fails to come up. And because this book of his seems to have captured some of the essence of the man himself I recommend it most heartily to everyone who enjoys a good autobiography.

L.B.

SCHIZOPHRENIA—1677

Schizophrenia—1677. A Psychiatric Study of an Illustrated Autobiographical Record of Demoniacal Possession. By Ida Macalpine, M.D. and Richard A. Hunter, M.D., M.R.C.P., D.P.M. Pp. ix + 197. 12 Plates, some in colour. £6 10s. 0d. (The edition is limited to 750 copies.) London: William Dawson & Sons Limited. 1956.

Contents: Acknowledgements. *Part I.* Introduction. Historical Review of the Contribution of Psychoanalysis to Psychiatry. Psychoanalytic Theory of Psychosis. *Part II.* The Story of the Manuscript. The Trophy of Mariazell. Notes. *Part III.* Freud's Analysis. The Evolution of the Devil. Haizmann's Devil. 'Ex Morte Ptarenis'. Homo sexuality and Castration Anxieties. Procreation Fantasies

and Sex Confusion. Course of Illness. Haizmann and Schreber. *Part IV.* Libidinal Conflict and Delusions. Hysteria: 'Psycho-Neurosis'. Hypochondriasis: 'Actual-Neurosis'. Paranoia: 'Narcissistic Neurosis'. The Case of Miss Anna O. Classification. Psychotherapy. Change of Sex and Procreation Fantasies. *References. Appendix. Index.*

This fascinating book, beautifully printed and illustrated, is from an edition limited to 750 copies. It is the second of the Psychiatric Monograph series published by William Dawson and Sons. The collection of material was supported in part by a grant from the Wellcome Trustees. All those who are interested in psycho-pathology and in the history of Medicine and of Art are indebted to the Wellcome Trustees and to the industry of the editors.

The book reprints the autobiography of a 17th century painter Christoph Haitzmann who suffered from schizophrenia and subsequently recovered to spend the rest of his life in a monastic order. In his illness he believed that he had sold his soul to the devil. His first cure followed exorcism by the Church. An early relapse was again treated by exorcism with apparently a permanent readjustment to life.

The artistic interest to the book lies in the reproduction of Haitzmann's eight visions of the devil, which give a valuable pictorial insight into the experiences of the mentally disturbed.

In the introduction it is stated: 'Quite apart from its intrinsic value, this manuscript has exerted an important and direct influence on present-day psychiatry, because it provided some of the fundamental evidence for the psycho-analytic theory of psychosis. In 1923 Freud made it the subject of an analysis under the title "A Neurosis of Demoniacal Possession in the Seventeenth Century". He believed that the possessed painter's illness confirmed, and so consolidated, the psycho-analytic theory of paranoia'.

J.F.B.

REFRESHER COURSE

Refresher Course for General Practitioners. Specially Commissioned Articles from the British Medical Journal. Third Collection April 1952 to September 1953 (Fully Revised). Pp. xvii + 548. Illustrations. 25s. net. London: British Medical Association. 1956.

Contents: Psychiatry Treatment and the Law (Alexander Kennedy). Treatment of Gonorrhoea (T. E. Osmond). Medico-Legal Aspects of Abortion (Donald Teare). Heart Block (Geoffrey Bourne). Treatment of Burns (J. P. Bull and D. M. Jackson). Gall-Bladder Disease (Harold C. Edwards.) Rheumatoid Arthritis (W. S. C. Copeman). Fractures in the Aged (Richard H. Metcalfe). Serum Reactions (L. J. M. Laurent and H. J. Parish). Exposure to Heat and Sunlight (B. G. Maegraith). Quarantine and Isolation (R. E. Smith). Lacerations of the Hand (R. G. Pulvertaft). Rheumatic Fever (C. B. Perry). Peptic Ulcer (C. F. W. Illingworth). Resuscitation after Drowning (E. J. Gordon Wallace). Anaesthetic Explosions (C. F. Hadfield). Surgical Relief of Pain (Lambert Rogers). Glandular Fever (Sir Henry Tidy). Irregular Vaginal Bleeding (Malcolm Donaldson). Nephritis (Robert Platt). Disseminated Sclerosis (J. W. Aldren Turner). Interpretation of Wounds (F. E. Camps). Typhus Group of Fevers (R. Lewthwaite). Vertigo (Terence Cawthorne). Intestinal Worms (A. W. Woodruff). Diagnosis of Smallpox (Thomas Anderson). Abdominal Hernia in Childhood (Denis Browne). Scabies and Lice (Godfrey Bamber). Acute Osteomyelitis and Septic Arthritis (Norman Capener). Mental Deficiency (Brian H. Kirman). Haemophilia (J. V. Dacie). Head Injuries (D. W. C. Northfield). Prevention and Treatment of Tetanus (Leslie Cole). Antenatal Care (W. C. W. Nixon). Chest Pain and Pleural Effusion (Howard Nicholson). Miscarriage (John Beattie). Tachycardia (Crighton Bramwell). Medical Aspects of Air Travel (Sir Harold E. Whittingham). Convulsions in Childhood (Bernard Schlesinger). Recurrent Boils (S. T. Anning). Prolapse (A. J. Wrigley). Late and Latent Syphilis (G. L. M. McElligott). Injuries to the Back (E. A. Nicoll). Anticoagulant Therapy (H. Payling). Hysteria (I. R. C. Batchelor). Carcinoma of the Breast (H. J. B. Atkins). Apoplexy (F. J. Nattrass). Treatment of Accidental Poisoning (John Glaister). Death Certification (W. P. D. Logan). Intermittent Claudication (J. B. Kinmonth). Myxoedema (A. W. Spence). Breast-Feeding Difficulties (F. Charlotte Naish). Pyelitis in Children (A. V. Neale). Leucorrhoea (C. S. Russell). Scrotal Swellings (R. J. McNeill Love). Indigestion in Childhood (Norman B. Capon). Cortisone and Corticotrophin (C. L. Cope). Spectacles (J. H. Doggart). Subacute Bacterial Endocarditis (Ronald V. Christie). The Anxiety-State (E. A. Bennett).

This is the third series to be published in book form of the B.M.J. Refresher Course for General Practitioners; this series was published between April 1952 and September 1953. The articles were specially commissioned, and each is written by an expert. They are short, requiring 20 to 30 minutes' reading, and are very suitable for filling in an idle half-hour. Those who have already read the articles in the B.M.J. will be glad to renew their acquaintance without having to chase them through numerous numbers of the Journal.

The articles are short, pithy and to the point, and they have been revised and brought up to date by their authors for the purpose of this publication. What is particularly pleasing is that they are all within the scope of the general practitioner.

The articles, besides being authoritative, are eminently readable. Inevitably, of course, the more interesting subjects have been dealt with in the first two volumes.

A cumulative classified contents list of all three books in the series adds to the usefulness of the volume, and may lead those unacquainted with the previous two volumes to enquire whether they are still in print.

F.R.L.

PYE'S SURGICAL HANDICRAFT

Pye's Surgical Handicraft. Seventh Edition Fully Revised. A Manual of Surgical Principles, Minor Surgery, and Other Matters connected with the Work of Surgical Dressers, House Surgeons and Practitioners. Edited by Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.R.S. (Edin.). Pp. ix + 800. 860 Illustrations. 52s. 0d. Bristol: John Wright & Sons Ltd. 1956.

Contents: I. The Arrest of Haemorrhage. II. The Treatment of Shock. III. Resuscitation. IV. The Treatment of Burns and Scalds. V. Bandages and Bandaging. VI. Bandages and Bandaging (continued): Adhesive Plaster Technique. VII. Hollow-needle Technique in Injection Therapy. VIII. The Administration of Sera. IX. Establishing and Maintaining Fluid and Electrolytic Balance. X. Blood Transfusion. XI. The Clinical Use of Anticoagulants. XII. Antibiotic Therapy. XIII. Sulphonamide Therapy. XIV. Pre-operative Medication and Basal Narcosis. XV. General Anaesthesia. XVI. Intravenous Anaesthesia. XVII. Examination of the Urine. XVIII. Preparation for Operation. XIX. Preparation of the Diabetic Patient for Operation. XX. Transnasal Gastric, Duodenal, and Intestinal Intubation: Technique and Uses. XXI. The Administration of Enemata. XXII. Assisting at Operations. XXIII. Repair of Operative Incisions. XXIV. Methods of Dressing and Draining Wounds: Factors in Wound Healing. XXV. Analgesics, Sedatives, and Hypnotics. XXVI. Post-operative Pulmonary Complications. XXVII. Post-operative Breathing and Other Exercises. XXVIII. The Management of Head Injuries. XXIX. The Management of Hare-lip and Cleft Palate Cases. XXX. The Management of Thyroid Cases. XXXI. The Management of Surgical Thoracic Cases. XXXII. The Management of Oesophageal Cases. XXXIII. The Management of Gangrene of a Limb, Threatened or Actual. XXXIV. The Management of Gastric Cases. XXXV. The Management of Gall-bladder and Pancreatic Cases. XXXVII. Ileostomy and its Management. XXXVIII. Renal Function Tests. XXXIX. The Management of Renal Cases. XL. Catheters and Catheterization. XLI. The Management of Bladder and Prostatic Cases. XLII. The Management of Rectal Cases. XLIII. The Management of Some Complications after Abdominal Operations. XLV. The House Surgeon and the Radiological Department (I). XLVI. The House Surgeon and the Radiological Department (II). XLVII. The Use of X-ray Apparatus by House Surgeons and Practitioners. XLVIII. The House Surgeon in the Pathological Department. XLIX. Some General Principles in Minor Operations. L. Minor Operations on the Male Genital Organs. LI. Minor Ano-rectal Operations. LII. Gynaecological Procedures and Minor Operations. LIII. Minor Operations and other Procedures which concern the Feet. LIV. The Treatment of Certain Abscesses. LV. The Treatment of Carbuncles and Bed-sores. LVI. Infections of the Hand. LVII. Injuries of the Hands and Fingers. LVIII. The Treatment of Diseases of the Veins. LIX. Medical 'Operations'. LX. Vaccination. LXI. Lumbar Puncture and Allied Procedures. LXII. Operations upon the Tonsils. LXIII. The Nose and its Accessory Sinuses. LXIV. The Larynx. LXV. The Ear. LXVI. The Eye. LXVII. Emergency Dental Treatment. LXVIII. The Skin. LXIX. The Treatment of Syphilis and Chancroid. LXX. The Treatment of Gonorrhoea. LXXI. The Treatment of Bruises and Sprains. LXXII. The Treatment of Manipulation. LXXIII. Plaster-of-Paris Technique. LXXIV. Emergency Treatment of Wounds and Compound Fractures. LXXV. The Treatment of Wounds and Compound Fractures. LXXVI. General Principles in the Treatment of Fractures. LXXVII. Complications of Fractures. LXXVIII. Fractures and Dislocations of the Upper Limb. LXXIX. Fractures and Dislocations of the Bones of the Face, Spine, and Pelvis. LXXX. Fractures and Dislocations of the Lower Limb. LXXXI. The Treatment of Tuberculous Joints. LXXXII. The Treatment of Acute Poisoning. LXXXIII. Medico-legal Reports. LXXXIV. Assessment of Incapacity. LXXXV. The Relationship of the House Surgeon to his Chiefs, his Patients, and the Nursing Staff. LXXXVI. Hospital Administration. LXXXVII. Certification of Death and Reporting to the Coroner. LXXXVIII. Death Certification in Scotland. Appendix. Index.

This excellent *vade mecum* of practical information, which has served generations of house surgeons and practitioners since Lister's day, has been brought right up to date. With the help of numerous contributors, each of whom is an expert in the field concerned, Hamilton Bailey has included in the 1956 edition references to all recent major advances in Surgery and Surgical Physiology. This edition contains a very large number of additional illustrations of technical procedures, which is a big improvement. The field covered is immense, with the result that the book has perhaps become too large, but Surgery has become so vast a subject that this cannot be avoided without detracting from the value and purpose of the volume. No other criticisms are offered.

The book is strongly recommended as a most valuable practical guide for all practitioners, house surgeons and medical students. Every surgeon should be familiar with its contents and specialists in other branches should benefit greatly by reading it.

J.H.L.